Vol. 67, No. 9 Established 1884 September 1950

ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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E. B. TREAT & CO., Inc., *Publishers*, 45 East 17th Street, NEW YORK. 3
Yearly Subscription $6.00 (Foreign $6.75); Single Copy, $1.00
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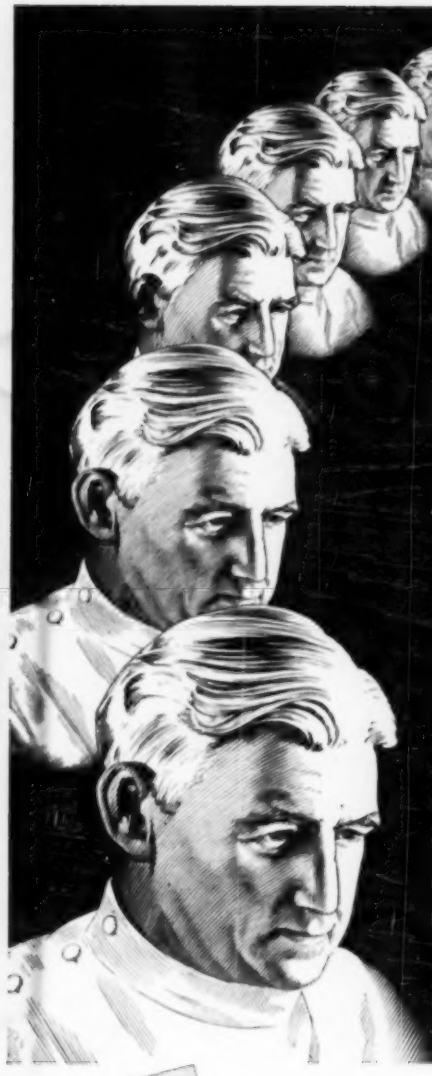

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Business Communications address to E. B. TREAT & Co., INC., 45 East 17th Street, New York 3

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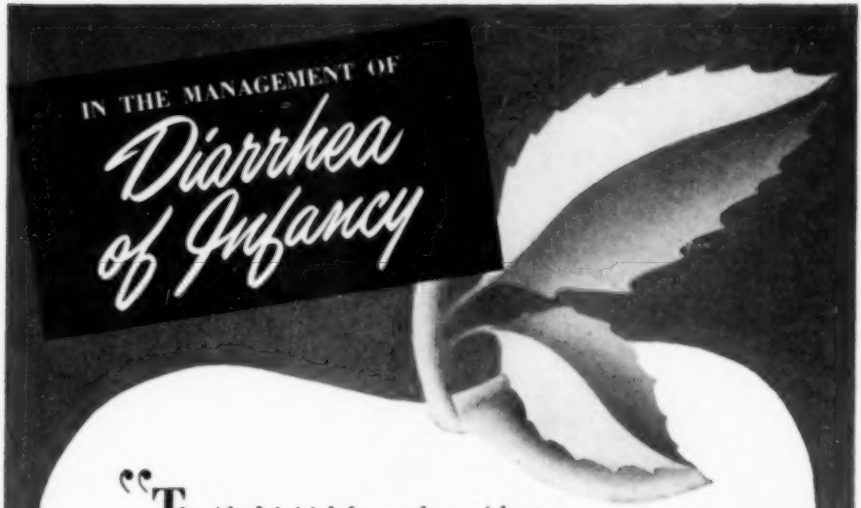

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1. O'Keefe, E. S.: *Am. Jour. Dis. Child.*, 76:616, Dec., 1948.

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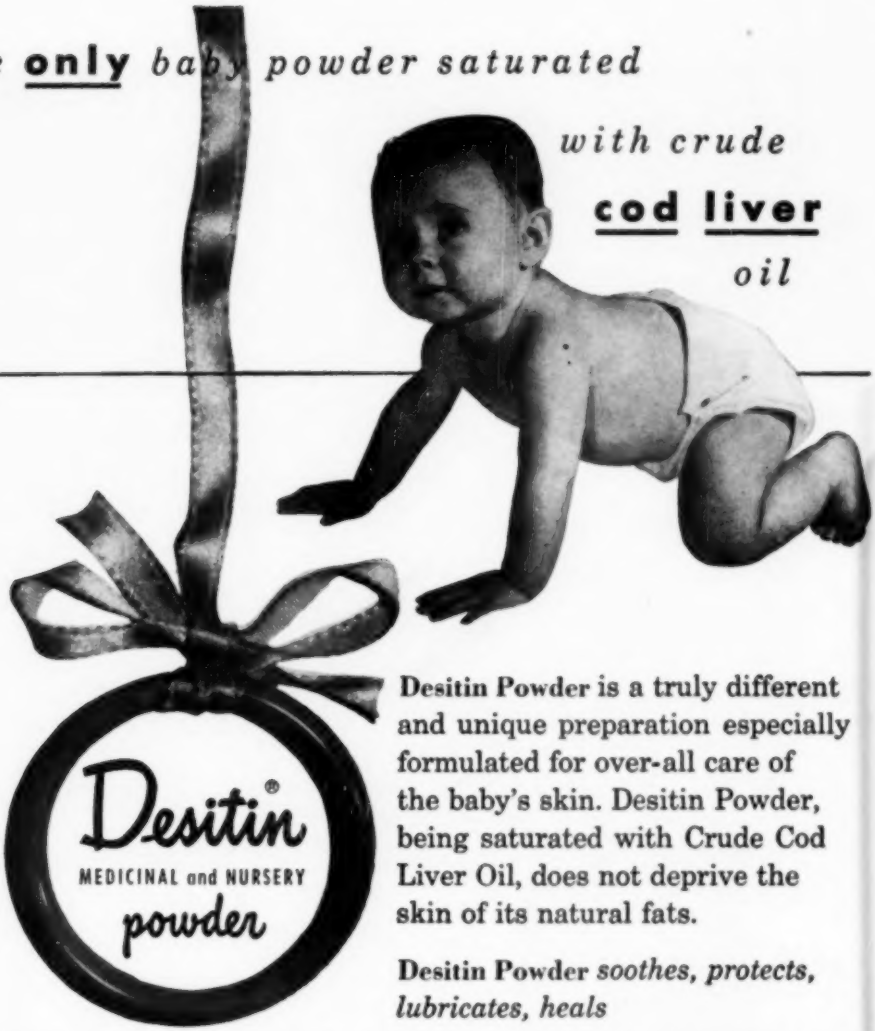

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Cullick, L. and Ogden, H. D.: J. South Med. Assn. 43: No. 7, July 1950

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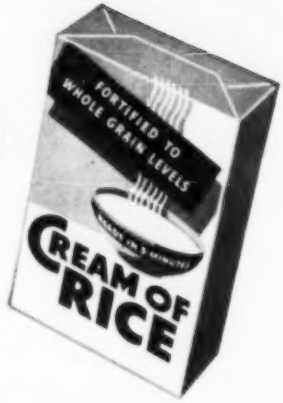

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HERPES ZOSTER IN THE NEWBORN ASSOCIATED WITH CONGENITAL BLINDNESS

REPORT OF CASE

CLEMENT E. COUNTER, M. D.
Long Beach, Calif.
AND
BERNARD J. KORN, M. D.
San Pedro, Calif.

The purpose of this report is to record the occurrence of herpes zoster in a very young infant. This disease, so often seen in adults, is infrequent in infants. Only seven cases have been reported in the newborn. The youngest child to have this disease is reported by Kolb[1], who explained the presence of symmetric scars on a newborn infant's face by the intrauterine development and healing of herpes zoster. Trausch[2] reported the case of a child born with unilateral herpetic lesions having a segmentary distribution. Freud, Rook and Gurian[3] reported herpes zoster beginning in an infant 3 days old. Bonar and Pearsall's[4] report was of an infant 4 days old. Bohn, quoted by Lower[5], in 1869, is said to have reported a case of this disease which began in a child 4 days old. Boix[6], of Montevideo, reported two cases of herpes zoster in infancy. One of these cases began in an infant 4 days old and the other was 4 months old. Katayama's[7] report of herpes zoster in a newborn infant began in a child 12 days old.

CASE REPORT

B. H. was a girl born September 29, 1947 by caesarean section. The mother insisted that she was one month overdue at the

time of operative delivery but the child weighed 6 pounds 14 ounces. The distal femoral epiphyses, which are usually united at full term birth, were ununited. It is probable that the infant was born 2 to 4 weeks premature.

On September 30, when the child was 20 hours old, the first patch of vesicles appeared in the right axilla. Other patches appeared until there were lesions on the posterior part of the neck from the third cervical to the third thoracic vertebrae. There were many groups of vesicles on the right shoulder and downward on the right side of the back over the scapula. Other groups were on the anterior part of the right shoulder and downward over the clavicle to the fourth rib. Other similar lesions were on all surfaces of the right arm and forearm and hand. There were right axillary lesions and palm lesions. No lesions were on the left side of the body.

When the infant was 7 days old, groups of vesicles were confluent and many had their central portion excoriated. Boric acid solution compresses were instituted as the only local application. Healing progressed readily through 3 weeks and she was discharged from the hospital with 3 small crusts and some pink patches remaining to show evidence of the vesicular skin disease. She weighed only 1 ounce less than at birth.

Laboratory examinations of the infant's mother just before surgery showed 3,250,000 erythrocytes, 68 per cent hemoglobin, 9400 leukocytes of which 77 per cent were neutrophiles and 23 per cent were lymphocytes. The serological tests for syphilis were negative.

The infant gained well at home. When she was 4 months old she weighed 11 pounds 1 ounce and was 22 inches long. She had begun to show signs of blindness at the various examinations between 4 months and 8 months of age. She did not smile and she did not play with her hands. She did not fix objects with her eyes nor did she follow a light with her eyes. She did not blink when the examiner's hand was thrust close to her eyes. Consultations with pediatrician and ophthalmologist resulted in no definite diagnosis of the kind of blindness except that it was of central congenital origin.

Contact with the infant was lost when she was approximately

10 months old. It is known that she died when about one year of age following the peculiar circumstance of having been abandoned on the steps of a public building. Autopsy was not done.

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7. Katayama: Japan. Ztsch.f.Dermat. & Urol., 43: 145, 1938.

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PERTUSSIS IMMUNIZATION. (Medical Journal of Australia, Sydney, *2*: 807, Dec. 3, 1949). Experiments by Gray in mice and rabbits showed that 24 hour Bordet-Gengou cultured phase I Haemophilus pertussis vaccine is highly protective for mice when given parenterally and stimulates the formation of protective antibodies and agglutinins against phase I Haemophilus pertussis in rabbits. The same vaccine, when given to mice orally, stimulates a lower but significant degree of protection which apparently cannot be greatly improved with even twenty-fold doses given by this route. Oral administration to rabbits did not stimulate the formation of agglutinins or protective antibodies. A proprietary dissolved oral vaccine dispensed for prophylactic and therapeutic control of whooping cough failed to produce significant immunity in mice when given orally or parenterally under the experimental conditions employed. It also failed to stimulate protective antibodies or agglutinins when given by either route in rabbits. The serums of 3 human volunteers to whom the dissolved oral vaccine was given were compared with the serum of a subject who was naturally immune to pertussis as the result of clinical infection followed by prolonged laboratory contact with Haemophilus pertussis phase I. The dissolved oral vaccine also failed to stimulate protective antibodies or agglutinins in the human volunteers. There is nothing in these results to suggest that effective immunization against pertussis infection is likely to be achieved with oral administration of any of the existing vaccines.—*Journal A.M.A.*

POISONING AS THE CAUSE OF POLIOMYELITIS

RALPH R. SCOBEY, M.D.

SYRACUSE, N. Y.

Poliomyelitis research, for the past 42 years, has been directed almost exclusively along only one line of investigation—the virus theory—whereas investigation of cause of other diseases, where progress has been significant, has *included* virus theories but also has investigated all other possibilities as well. That there are causes for poliomyelitis other than a virus has been pointed out repeatedly.

Concepts regarding the nature of many diseases are changing and with these changes progress for the betterment of human welfare is inevitable. Poliomyelitis investigations, unfortunately, have not shown the same degree of progress noted in other fields of medicine. Essentially most of the questions regarding the epidemiology of this disease, that were unanswered half a century ago, still remain unanswered or are explained hypothetically. Dauer[1] (1938), epidemiologist, has said truthfully, and it is applicable today: "It seems rather remarkable in spite of all the time and effort spent in poliomyelitis studies during the past 20 years so little information in the epidemiology of the disease has been produced."

There is one theory, which will be considered in this report, besides the virus theory of cause of poliomyelitis, that stands out predominantly but never has been adequately investigated. This "poison theory", although emphasized for centuries, has been ignored for the past half century. Ironically, the word "virus" is derived from the Latin meaning poison, and when physicians of past generations referred to a virus they definitely meant a poison. Although it has been assumed that poliomyelitis is a modern disease, it will be shown in this report that merely the name is modern; the disease has been familiar to physicians for centuries.

POISONING AS A CAUSE OF PARALYSIS

Boerhaave[2] (1765), Germany, states: "We frequently find persons rendered paralytic by exposing themselves imprudently to quicksilver, dispersed into vapors by the fire, as gilders, chemists, miners, etc., and perhaps there are other poisons, which may pro-

duce the same disease, even externally applied." His reference to anapoplectic waters, antiparalytics, etc., to be found in the Materia Medica, indicates that paralysis was probably not uncommon in those days.

Boerhaave states: "Hippocrates sometimes understood by the word apoplexy, a palsy of a particular part of the body, as it is also observed by Areteus and Aegineta, for he applied the word apoplectic to the leg. There are several places in Hippocrates where the apoplexy is evidently taken for palsy of a particular part. But if this disease is produced by poisons, there is no remedy hitherto discovered to cure it."

Although alcoholism has without doubt been known for centuries, the earliest account of alcoholic paralysis is said to have been given by Jackson,[3] of Boston, in 1822. Alcoholic paralysis obviously cannot be considered a new disease merely because it was described for the first time by Jackson in 1822. Very little attention was given to Jackson's observations until they were confirmed in 1849 by Magnus Huss,[3] of Stockholm. In 1878, Wilks,[3] of England, drew attention to the frequency of paraplegia in alcoholics. Alcoholic paralysis was accurately described by Lancereaux[3] in 1881.

Cooke[4] (1824) states: "Among the exciting causes of partial palsies we may reckon the poison of certain mineral substances, particularly of quicksilver, arsenic and lead. The fumes of these metals, or the reception of them in solution into the stomach, have often caused paralysis."

Colton[5] (1850) mentions the case of a patient who swallowed some arsenic accidentally and was admitted to the hospital. The primary effects of the poison had been successfully combated with proper remedies, but seven days afterward he became paralyzed.

Landry[6] (1859), who first described the acute ascending paralysis that bears his name, ascribed it to an intoxication. A similar view was entertained by Hayem.[7] Westfall[8] (1876) considered that some form of intoxication is the actual and final cause of acute ascending paralysis. Walton[9] (1895) emphasized that Landry's paralysis is an acute toxic disease.

Packard[10] (1879) reported the simultaneous occurrence of poliomyelitis in a brother and sister. As a possible explanation for its simultaneous occurrence, he suggested the action of toxins re-

sulting from their absorption from the digestive tract. He says that "the action of toxins could produce the anatomical changes seen in this disease is not at all unlikely in view of our knowledge of their destructive power in other parts as well as the central nervous system."

Vulpian[11] (1879) experimentally produced paralysis of the extensors of a dog by lead poisoning. The lesions, consisting of colloid degeneration and cell atrophy of the anterior horn cells of the spinal cord, were pronounced by Vulpian as poliomyelitis. Adamkiewicz[12] (1879) reported two parallel cases, one of poliomyelitis and one of lead poisoning.

In 1881, Popow,[13] of St. Petersburg, published an essay upon the pathological anatomy of arsenical paralysis as produced artificially in animals. The work of Popow was carried out under the guidance of the distinguished neurologist and microscopist, Professor Mierzeyewski. Popow concluded that arsenic, even in a few hours after its ingestion, may cause acute central myelitis or acute poliomyelitis.

Mills[14] (1883) describes seven cases of arsenic poisoning, strikingly similar to acute poliomyelitis, resulting from the eating of pumpkin pie proved to contain arsenous acid. One outstanding case was that of a man, age 24 years, who became paralyzed in the arms and legs nine days after the ingestion of the poisonous pie.

Jacobi[15] (1886), an outstanding authority on infantile paralysis, states in her classical review of this disease: "The acute ascending paralysis of Landry has been said to strinkingly resemble the effects of poison. It may be noticed that the occurrence of spinal accidents after ordinary infectious diseases, as scarlatina and measles, should as well indicate that a specific agent proper to itself was at least not essential to its development. The influence of exposure to cold seems to have been demonstrated, must probably be interpreted, as effective by means of some poison generated in the organism when cutaneous secretion, exhalation or circulation has been suddenly checked."

Starr[16] (1887) points out that all alcoholic drinks are not prone to produce paralysis. "It is the spiritous liquors—brandy, whisky, gin, and rum, and the liqueurs, absinthe, vermouth, etc.—which are dangerous. . . . .Charcot goes so far as to say that muscular sensitiveness, associated with motor paralysis, is pathognomic of alcoholism."

"The course of alcoholic neuritis is quite uniform," Starr says. "After a sudden onset the symptoms rapidly advance to a high degree, which is reached in a week or two from the beginning of the paralysis or ataxia. Then they may increase further and cause death by respiratory paralysis. . . . In a few cases the muscles become contracted, and permanent deformities, only to be overcome by long-continued massage or operative measures, develop."

Putnam[17] (1895) states: "It is not impossible that we group, clinically, under the name poliomyelitis several affections which might in strictness be separated, or in other words, that several different poisons are liable to affect the anterior horns of the spinal cord, exciting results which, though in the main alike, may differ in detail. . . . Although the idea is generally accepted that there is a toxic cause for poliomyelitis, no one has strongly affirmed this disease is of bacterial origin." Putnam points out that the abrupt onset and rapid course of Landry's paralysis point definitely to some toxic cause.

Luisada[18] (1895) reported that the theory of Mya is that the origin of infantile paralysis is in a septic or toxemic process of gastro-intestinal origin.

Caverly[19] (1896), who investigated and reported an epidemic of poliomyelitis which occurred in 1896 in Vermont, says: "That the disease occasionally prevails epidemically suggests a specific poison, a definite toxin. . . . There was a general absence of infectious disease as an etiological factor in this epidemic; the element of contagion does not enter into the etiology either. I find but a single instance in which more than one member of a family had the disease, and it usually occurred in families of more than one child, and, as no efforts were made at isolation, it is very certain it was non-contagious."

Thomas[20] (1898), writing on Landry's paralysis, states: "Inasmuch as nerve cells react in much the same way to various poisons, further research will probably show that in these cases micro-organisms are not always present, but the intoxication may be produced through faulty metabolism or by the absorption of poisons from without. That toxic substances may act in a selective manner, acting only on motor neurones, is difficult to explain, although we are not without other instances of the same action, for example, the almost pure motor trouble in lead paralysis."

Stieglitz[21] (1900) experimented with lead poisoning in 36 animals, and in the guinea pig found characteristic changes with destruction in the anterior cornua with cellular infiltration. Paralysis was present and death occurred in 24 hours.

Onuf[22] (1900) reported the case of a painter with flaccid paralysis of both legs, in whom the autopsy showed lesions characteristic of poliomyelitis.

Obrastoff[23] (1902) reported a case of acute poliomyelitis resulting from arsenic poisoning.

Gossage[24] (1902), writing on infantile paralysis, says: "The nerve cells or fiber may be acutely disabled by the action of some poison circulating in the blood, and it is possible that such a poison would only temporarily impair their functions or so seriously damage them that recovery would be impossible."

Phillippe and Gauthard[25] (1903) reported a case of anterior poliomyelitis from lead poisoning.

Ham[26] (1905) calls attention to the fact that formalin was common in the milk supply of Brisbane, Australia, and records an instance of an adult who was suddenly attacked with paralysis after swallowing a small amount of formalin.

Collins and Martland[27] (1908) reported a case of poliomyelitis in a man, 38 years of age, which resulted from the use of potassium cyanide as a silver polish. The illness began with diarrhea, followed by headache and pain and stiffness in the back of the neck. About eight days after the onset of illness, he became paralyzed. In discussing Collins and Martland's paper, Larkin stated that he had seen one instance of this disease following potassium cyanide poisoning.

Collins and Martland poisoned several rabbits with potassium cynaide and found pathological lesions in the spinal cord identical with those found in cases of poliomyelitis.

Lovett[28] (1908), describing an epidemic of poliomyelitis in Massachusetts in 1907, and after reviewing the literature on experimental poliomyelitis, states: "The injection experiments prove that certain metallic poisons, bacteria and toxins have a selective action on the motor cells of the anterior cornua when present in the general circulation; that the paralysis of this type may be largely unilateral; that the posterior limbs are always more affected than the anterior; and that the lesions in the cord in such cases do not differ from those in anterior poliomyelitis."

Wynkoop[29] (1916) suggested that poliomyelitis is caused by poisons produced within the body by perverted functions of organs or poisons introduced within the body from without by food, drink or inhalation.

Gougerot[30] (1935) reported that during arsenical therapy for syphilis, poliomyelitis developed in two patients and lethargic encephalitis followed by Parkinson's disease in one.

Lœnhardt et al.[31] (1931) described acrodynia in the course of three cases of acute poliomyelitis. Some authorities have considered acrodynia to be caused by a poison, i.e., spoiled corn, mercury, arsenic, ergotism, etc. Elmore[32] (1948) reported two cases of this disease following the ingestion of mercury, and Warkany[33] found that the urine of several patients with acrodynia contained appreciable amounts of mercury. Mayerhofer[34] (1939) reported that infantile acrodynia may immediately follow certain forms of atypical poliomyelitis, especially encephalomyelitis.

Hammes and Frary[35] (1936) described the acute onset and rapid development of motor paralysis within two weeks after a simple laparotomy in three members of one family. It is possible that the anesthetic may have been a contributing factor.

Martini[36] (1937) suggested a possible relationship between poliomyelitis and the toxins of the ascaris. He pointed out that ascaris toxins are neurotropic, especially for the spinal cord and meninges.

Wolter[37] (1938) states: "According to the newer Pettenkofer school, epidemic poliomyelitis is due to a poisonous substance with special neurotropism which develops from chemical processes in an unclean ground, like a fermentation process, in essential specificity. This soil exhalation affects the human organism by way of the respiratory organs." It is felt that this is a gaseous substance.

Gelperin[38] (1950) felt that many ills are the result of poisoning. He considered that large numbers of what are believed to be poliomyelitis cases are in reality poisoning cases. J. G. Townsend,[39] Medical Director, U. S. Public Health Industrial Hygiene Division, stated that one of the symptoms of parathion poisoning is extreme muscular weakness and that it is possible that some sicknesses mistaken for poliomyelitis may have been caused by eating fruits or vegetables bearing parathion residue. "Some medical authorities see more than a coincidence in the distribution and

use of parathion for the first time in 1948, and the epidemics of polio in sections of the country last year"[40]

Matson[41] (1950), writing on "Hidden Enemies in the Garden" points out that thousands of cases of poisoning occur each year due to noxious plants. "People occasionally become ill," she says, "by eating partridge meat, and investigators have advanced the theory that the flesh of the birds is poisoned by the seeds and berries of poison plants, particularly the laurels, whose toxic constituents always lodge in the flesh of their victims."

In describing poisoning by snakeroot, the active principle of which is tremetol, she states: "Some physicians have even expressed the opinion that mysterious outbreaks of so-called 'summer flu' in the late summer are often due to milk contaminated with tremetol."

FOOD POISONING AS A CAUSE OF PARALYSIS

The finger of suspicion has for centuries been pointed toward food as the cause of paralytic afflictions occurring during the season when poliomyelitis epidemics are known to occur.

Cholera morbus, first described by Sydenham[42] in *1669*, which appeared in epidemic form in the summer and autumn, and manifested itself chiefly by gastro-intestinal complaints corresponds with the diseases that now occur during an epidemic of poliomyelitis. Later writers designated these complaints not only as cholera morbus but as summer complaint, summer and autumnal diseases, diseases of the season, cholera, remittent fever, intermittent fever, etc. Cholera morbus was not infrequently followed by paralysis as indicated by Boerhaave,[2] Rush,[43] and others. The primary reaction in cholera morbus, as pointed out by Chapman,[44] (1883) is in the gastro-intestinal functions and thence to the spinal marrow and brain. Significantly, Medin[45] (1895) reported, during an epidemic of poliomyelitis, that epidemic infantile cholera was especially severe during the months of August and September. Boerhaave[2] points out that he learned from Dr. William Bull, a practitioner in America, that this colic is so frequent in the south parts of America that it may almost be reckoned as endemic there. Roberts[46] (1858) speaks about the palsy that follows endemic colic of certain countries.

The cause of cholera morbus was attributed by writers since

Sydenham's time to unripe fruits and vegetables, fish, shellfish, stagnant water, miasm, etc. An editorial in the Boston Medical and Surgical Journal for 1887[47] states concerning cholera morbus: "Certainly, in the present state of medical science, there is not sufficient warrant for classing this so-frequent malady of the hot weather among the infectious or bacillary diseases. . . . The experience of practitioners generally corroborates the above view, that attacks of cholera morbus are ordinarily caused by some fermentative change in the ingesta, and here it is principally the quality of the ingesta that is operative, unripe fruits or vegetables, and food that is relatively indigestible being most likely to produce an onset. The patient, on being interrogated, will generally refer his sickness to some indiscretion in the quality of the food which he has eaten. . . . It will not do, also, to omit the influence of drinking water."

Cruveilhier[48] (1833), as well as others, pointed out that the patient with cholera morbus presents the symptoms of poisoning, a fact which he says was noted by Morgagni, "and almost every writer since his time."

"In the 16th century during epidemics in which no symptom or sign of polioencephalitis, poliomyelitis, or cerebrospinal meningitis was lacking, ergot, bad bread, sour apples, mushrooms and the like were mentioned as causes.

"In epidemics in 1729-30, the common people accused the weather, the grains, the bread, and unripe fruits. It was noted in some of these epidemics that domestic fowls were at the time affected by what is now called 'limber neck.'

"In 1820, Justin Kerner, of Weimsberg, attributed outbreaks, clinically identical with polioencephalitis, to the eating of Swabian sausages. It was also long the custom in Germany to incriminate ergot of rye as the cause of certain acute, epidemic maladies, characterized by respiratory and gastro-intestinal catarrhs. These maladies frequently entailed permanent paralysis and withering of the limbs; they affected the young at least as frequently as (according to some authorities more frequently than) their elders. This was the affection or group of affections known popularly in Germany as Kriebelkrankheit and otherwise; called raphania in Sweden by Linneus since he thought it due to radish-seeds and not to ergot; and (about 80 years ago) replaced in the minds

and textbooks of Swedish physicians by cerebro-spinal meningitis, polioencephalitis, and poliomyelitis.

"In 1830, an outbreak of raphania was heard of again in Germany. In Wurtemberg botulism and encephalitis were described within a few miles of each other in 1834 and 1835. *In the same years and in the same kingdom, Heine noted his first case of infantile paralysis. In* 1835, *Badham's cases of the same disease were reported in England, and also the publications by Ryan, a long forgotten English physician, described inter alia, cerebral and spinal forms in children.* In Wurtemberg, in 1850-51, there were again outbreaks of botulism.

"Raphania was flickering out in Sweden in the fifties for it then and there was beginning to be called cerebro-spinal meningitis; but at the same time southwestern Germany and Bavaria, with Hesse-Nassau, again suffered an extraordinary medley of outbreaks of botulism, raphania, localized influenzas, encephalitis and the like diagnostic entities. The last trace of raphania and epidemic psychopathy in Sweden (1869) coincides with the first recognized outbreak of poliomyelitis in Norway. In 1895, Van Ermengem observed the Belgian outbreak which was clinically polioencephalitis but declared to be botulism."[49]

Barton[29] (1827) wrote an account of the poisonous and injurious effects of the honey of North America which derived these poisonous qualities from the flower of certain plants (laurel; kalmia hirsuta, andromeda mariana; wild honey-suckle; datura stramonium). Paralysis was one of the sequelae of this honey poisoning.

Tobey[51] (1843) described the development of a partial paralysis of the face of his son, 12 years of age. This paralysis occurred while he was passing the summer in New Hampshire with his relatives. For two weeks prior to his illness, he had partaken plentifully of baked apples and milk.

The failure of the people's principal food—the potato crop—in Ireland in 1817 and 1849, caused them to eat spoiled potatoes and to partake of other foods to which they were unaccustomed. The illness that resulted was often designated epidemic fever, and paralytic manifestations were commonly observed as sequelae. "It invaded the rural districts in each year, in the purest localities, and quite apart from infections or contagion," one writer pointed out.

Robert Law[32] stated that the effect in some cases resembled that of a strongly concentrated poison. He says: "The individual in many cases seemed to suffer intensely as in the severest cases of rheumatism. These pains were more or less general in different patients. In some they affected the back of the head and neck; in some they only ran down the leg; while in others they spread themselves through the whole body, and, embracing the sides, imparted the sensation of painful constriction. The nape of the neck and across the groins were the points of the spine to which the patients most frequently referred their pain. When it occurred in the former position, which it did in many cases, we regarded it with some apprehension, from its being the starting point of that formidable disease, cerebro-spinal meningitis, into which we are sure it would have passed had it not been arrested by treatment.

"The cases of fever which constitute the subjects of the foregoing observations, for the most part, came from rural districts of Donnybrook and Castleknock. . . . From June 28, 1848, the wards were opened to receive patients and from this date to August 31st, 160 patients were received and treated, without a single death."

Purefoy[33] (1853), reporting on the same epidemic in Ireland, employs the name "remittent fever" for it. He says: "During the famine period in this country it is notorious that the want of food, its bad quality in many cases, the irregular or even excessive supply at some meals, as when Indian corn stirabout was supplied gratuitiously at eating houses; the long fasting and occasional gorging were factors."

"On its first appearance," Purefoy states, "the fever was characterized by suddeness of invasion, indicated by chills or rigors, pain of the head, occasionally extending to the back and limbs; in severe cases nausea and retching." He says there was a morbid affection of the cerebellum and spinal marrow, supposed to be spinal arachnitis, and characterized by severe pain along the spinal column, with rigidity of the dorsal muscles, flexure of the head backwards. The disease was differently denominated by different observers as famine fever and relapsing fever, he says. The disease, as it now occurs, he points out, is most frequently observed in the spring and autumnal seasons, chiefly affecting the young and middle aged.

An observation made by Purefoy, which appears to correspond with observations in cases of poliomyelitis made by Watson et al.[54] (1947) was a scanty reddish urine. This suggests the presence of porphyrin which was noted by Watson et al.

"Potato poisoning is of more frequent occurrence," Harris[55] (1928) says, "than is generally supposed. In the agricultural districts 'new' potatoes and those that are old and 'sprouting' are thought to be frequent sources of food poisoning. The toxic principle is said to be solanin."

The symptoms of potato poisoning in an outbreak of 67 cases with one death in Glasgow in 1917, studied by Wright, Harris, and Cockburn,[56] were chiefly those of gastro-enteritis, i.e., vomiting, diarrhea, colicky pains, headache, slight fever, and prostration. Partial paralysis occurred in some cases. It is significant that Colton[57] (1917), as well as Simonton[58] (1917) refer to the potato as an exciting cause of poliomyelitis.

Harley[59] (1863) exhibited specimens of the Calabar bean employed by the King of Calabar as a poisonous ordeal to determine the guilt or innocence of accused persons. Taken internally it produced fatal paralysis; small quantities produced partial paralysis.

In 1907, Emerson,[60] investigating an epidemic of poliomyelitis in Massachusetts, made a careful inquiry regarding the diet. No infant who was fed exclusively on the breast developed poliomyelitis. He found in six cases that fruit and berries had been a large item of the diet. In the cases of two infants, bananas and berries had been given in the diet in addition to breast milk. In one case of poliomyelitis the illness was attributed to the eating of large amounts of blackberries and blueberries. In 39 instances it was stated that food supplies were bought from fruit and vegetable peddlers and it was found that their carts served the town and country districts in their localities.

Milk, which at times is known to contain toxins resulting from poisonous plants eaten by cows while grazing, can cause illness in humans. Jubb[61] (1915) reported a milk borne epidemic of poliomyelitis, and several similar epidemics[62, 63, 64] have been reported since then that were traceable to milk. Walters[65] (1944) found on analyzing 100 unselected cases of poliomyelitis, that 68 patients had used raw milk for an indefinite time before their illness.

In 1916, Hart, Miller and McCollum[66] described changes in the anterior horn cells of pig's cords produced by means of diet. The authors decided they were dealing with a form of beriberi, not, however, due to a deficiency of vitamin B which was present in abundance in the wheat germ of the diet. The pathological condition was ascribed to an intoxication by the wheat germ. By supplementing the diet with alfalfa meal and meat scraps, the cell changes were prevented.

Réymond and Columbies,[67] in 1923, reported poliomyelitis following the ingestion of mushrooms. In 1924, Mac Guire[68] described a case, strikingly similar to poliomyelitis, of mushroom poisoning in a six-year-old boy. The mushrooms consumed were the ordinary edible variety as far as could be determined. Marcovitz and Alpers[69] (1935) reported two cases with toxic encephalitis following the ingestion of mushrooms. "Some authors," they state, "still insist on the presence of some virus as the explanation of this phenomena."

Mellanby[70] (1931) was able experimentally to produce and prevent degeneration of the spinal cord by diet. In some litters of puppies, when the diet contained much cereal and was deficient in fat-soluble vitamins, these changes were noted. Mellanby was able to produce nervous ergotism under certain conditions and found that slight alterations in the diet of a specific nature completely prevented the degenerative spinal cord changes, even when the same or larger amounts of ergot were eaten. Experiments carried out to test the action of rye germ on the spinal cord (minus the ergot) proved that this substance also contains the nerve toxin. He found that foods rich in vitamin A would prevent the ergot of cereal from producing the nerve toxin degeneration. It could also be prevented by carotene itself and by carrots which contain carotene. Mellanby found that it was impossible to produce spinal cord degeneration when the cereal of the diet consisted of yellow rather than white maize since the former furnished sufficient carotene. The latter, however, resulted in spinal cord degeneration.

Regarding vitamin A or carotene as a curative agent, Mellanby says: "There is definite evidence that the clinical condition tends to clear up rapidly under these conditions, though the degree of improvement naturally depends on the extent of the initial lesion.

The immediate effect is often astonishing even when the animal has been paralyzed for a long time. In a week or two the weak and even paralyzed hind legs become active once more . . . presumably further degeneration ceases on giving the additional vitamin A or carotene, and the fibers already showing degeneration are gradually restored."

Mellanby points out that the degree of degeneration in some tracts of the spinal cord varies to some extent from level to level, and degeneration may be evident in a tract at two levels in the cord whereas at an intermediate level little or no degeneration may be found in the corresponding area. He says: "The anterior columns are most affected whichever region of the cord is investigated."

Mellanby states: "Another point of interest is that in epidemics of nervous ergotism some members of a family may be attacked and others escape. The reason for this indiscriminate picking out of individuals may be related to the reserves of vitamin A in the livers of the individuals."

Reyes[71] (1945), during an epidemic of poliomyelitis in New York City, made observations that parallel those made by Mellanby in his experiments on animals. Reyes noted that 98 per cent of 84 children admitted to St. Francis Hospital suffereing from poliomyelitis had hyperkeratinization lesions of the skin due to vitamin A deficiency. The skin lesions appeared to be proportional to the severity of the poliomyelitis involvement. In the group of 84, all except two recovered completely; only two were unable to walk. The treatment consisted of a well balanced diet with the free administration of food containing vitamin A. "At the time of discharge the lesions of hyperkeratinization had almost disappeared in all the children, with the exception of the two above noted who failed to recover completely," Reyes says.

Stockman and Johnson[72] (1933), like Mellanby, found spinal cord degeneration in animals fed cereals when cereals constituted the bulk of the diet, chiefly including maize, rice, rye, wheat and oats. An acid obtained from these cereals, when administered by the stomach or hypodermically as a sodium salt, caused paralysis in many of the animals, including monkeys and rabbits. Different samples of cereal varied in toxicity and also in the effect on individual animals.

Franke[73] (1934) also found a toxin in certain cereals. Samples

obtained from farmers whose livestock had shown pathological symptoms exerted extremely toxic effects on white rats. The resistance of the rats was increased by the use of various adjuvants. The grains included corn, wheat, barley and emmer. In a number of the rats the hind legs were completely paralyzed. The toxin, Franke felt, was associated with the protein fraction of the cereal.

Chapman[74] raises the question of food poisoning to explain the epidemic of poliomyelitis in England in 1947, when he asks: "Is it not possible that the present prevalence of infantile paralysis may, in part at any rate, be due to some article in our restricted and modified dietary? In the Central Provinces of India there were after famine years widespread outbreaks of a form of spastic paralysis. This was caused by the consumption of the millet, Lathyrus sativa. In ordinary times this was only used as an adjuvant to the other grains, and it was only when used as a staple diet that the paralysis occurred. Is it not possible that in the modification of the austerity diet we may have introduced some article which may be causing this widespread epidemic."

Toomey and August[75] (1932) pointed out that some authors thought that poliomyelitis is a disease of gastro-intestinal origin which might follow the ingestion of foodstuffs. In 1933, Toomey and August[76] noted that the epidemic peak of poliomyelitis corresponds with the harvest peak of perishable fruits and vegetables. They called attention to the fact that the disease occurs only in those countries which raise the same type of agricultural products. Dr. C. W. Burhans, one of the colleagues of the authors, thought that green apples might be a factor in the etiology of poliomyelitis. Toomey et al.[77] (1943) point out that there is frequently a history of dietary indiscretions previous to an attack of poliomyelitis. They suspected that a virus could be found in or on unwashed fruit or in well water during epidemics of poliomyelitis. Every year for eight years, therefore, grapes, apples, peaches, and pears were collected from the vineyards and trees in Northern Ohio at the time of the ripening. In none of their studies was the so-called virus of poliomyelitis demonstrated when the washings of the fruit or the well water was injected into experimental animals. No chemical tests were made to determine, however, whether or not a chemical substance within the fruit or in the well water,

acting by oral ingestion to produce poliomyelitis, was present.

Draper[78] (1935) recorded a series of cases of poliomyelitis which he postulated originated from a Greek fruiterer. All of the cases were in contact with the Greek as business associates, relatives or customers, and there was nothing in the evidence to point to infection being carried by the Greek himself other than the fruit he supplied.

Leavell[79] (1936) suggested that the ingestion of uncooked fruits and water might be responsible for poliomyelitis.

Barber[80] (1938) reported four cases of poliomyelitis that developed simultaneously in a single house of a boarding school on the same day from the eating of strawberries. He says that the simultaneous onset of these cases resembled food poisoning. The seasonal and climatic incidence of poliomyelitis, he points out, agree closely with the seasonal increase in the consumption of fresh garden production. He says that the epidemiological distribution of poliomyelitis resembles food poisoning.

Chenault[81] (1941) noted that the history of poliomyelitis points to "a suggestive parallelism between a number of epidemics and the appearance of fresh vegetables and fruits."

Goldstein et al.[82] (1943) reported an epidemic of polioencephalitis at a naval training school among the cadets. The epidemic was explosive in character and involved over 100 persons. Epidemiological evidence suggested that some food served in the mess hall was the cause of the disease.

Kovar[83] (1945) noted the frequency of acute gastro-intestinal upsets at the time of epidemics of poliomyelitis in Nebraska in the years when severe drought and economic depression were prevalent. The symptoms and signs of many of his cases were similar to those of preparalytic poliomyelitis, but the patients had negative spinal fluids and recovered without paralysis. One case, a boy aged 13 years, had a diet for a few days prior to his sickness which consisted of foods of harvest that are capable of producing gastro-intestinal upsets. In another case, a boy, 9 years of age, had eaten a number of apples along with other fresh harvest foods the day before the onset of his illness. A third child, age 15 years, had been drinking large quantities of water and had had a very underbalanced diet for a week prior to his illness. Kovar considered the symptoms in the cases that he had observed as

gastro-intestinal in origin because the diets in each of his cases consisted of food capable in themselves of producing gastro-intestinal upsets. He says: "Because of the economic depression, proper food could not be purchased, and because of the drought proper food could not be obtained or raised."

Gebhardt and MacKay[84] (1946) found during an epidemic of poliomyelitis in Utah that 206 of 206 patients surveyed had one or two weeks prior to the onset of the disease eaten fresh fruits and vegetables. The authors found in Utah, New York and California, during 1943, that the cases of poliomyelitis paralleled the harvest peaks. Most of the multiple cases in families were found to have developed at the same time, suggesting means other than contact as the mode of spread.

An epidemic of poliomyelitis in the Middle East, investigated by van Rooyen and Kirk[85], in 1946, pointed toward uncooked food and vegetables as the cause of the disease.

Scobey[86] (1946) in an article entitled, "Food Poisoning as the Etiological Factor in Poliomyelitis," pointed out that the frequent reports of poliomyelitis following the ingestion of mushrooms, fruits, vegetables and milk could be attributed to the cyanide content of these foodstuffs. In a later report[87], he pointed out that water, under certain conditions, especially drought, could also contain this poisonous substance.

It is well-known by veterinarians and farmers that certain grasses, including Johnson grass, Sudan grass, millet, etc., affected by drought, contain a high percentage of cyanophore glucosides capable of producing hydrocyanic acid resulting in paralysis and death by respiratory paralysis of cattle. Leaflet 88, U. S. Department of Agriculture, entitled, "Poisoning of Livestock by Plants that Produce Hydrocyanic Acid" describes the effects of these plants on cattle. Cyanide is to be found as a glucoside also in plants that belong to the rose family which includes many of our common fruits and in members of the mustard family, including cabbage, cauliflower, etc.

Adamson et al.[88] (1949) reported an epidemic of poliomyelitis in the Arctic involving 78 Eskimos between the ages of 8 and 64 years. The explosive and devastating character of the epidemic strongly suggests food poisoning as the etiological factor. This fact is borne out by noting that there was a scarcity of food and

there was gorging of it when a supply was obtained; the Eskimos had little regard for the state of preservation of the food and preference for meat when it was "high"; the eating habits were irregular, with a minimum amount of preparation and cooking; no child under three years of age was afflicted with the disease since the Eskimo mothers nurse their babies until that age; and gastro-intestinal disturbances were more prevalent than usual during epidemics of poliomyelitis.

Barondes[89] (1949) points out that a study of the epidemiology of poliomyelitis shows a definite correlation with the harvesting of fruit and vegetable crops and to changes in climate, weather and humidity. The harvesting of such fruits as cherries, grapes, berries, apricots, etc. and the edible vegetables, as lettuce, radish, cucumbers, etc. usually from June to September corresponds with the period of poliomyelitis epidemics, he says.

PARALYSIS FROM POLLUTED WATER

Hippocrates[90], writing on "Airs, Waters and Places," makes some pertinent remarks which have been more or less ignored in the investigation of poliomyelitis. He says: "Whoever wishes to investigate medicine properly, should proceed thus: in the first place to consider the seasons of the year, and what effect each of them produces. Then the winds, hot or cold, especially as are common to all countries, and then such as are peculiar to each locality. We must also consider the qualities of the waters, for as they differ from one another in taste and weight, so also they differ much in their qualities. . . . These things one ought to consider most attentively, and concerning the waters which the inhabitants use, whether they be marshy and soft, or hard and running from elevated and rocky situations, and then if salty and unfit for cooking, and the ground, whether it be naked and deficient in water, or wooded and well watered, and whether it lies in a hollow, confined situation, or is elevated and cold; and the mode to which the inhabitants live, and what their pursuits, whether they are fond of eating and drinking to excess, and given to indolence, or are fond of exercise or labor, and not given to excess in eating and drinking. *For from these things he must proceed to investigate everything else.* For if one knows all these things well, or at least the greater part of them, he cannot miss

knowing, when he comes into a strange city, either the diseases peculiar to the place, or the particular nature of common diseases, so that he will not be in doubt as to the treatment of the diseases, or commit mistakes, as is likely to be the case provided one had not previously considered these matters. And in particular, as the season and year advances, he can tell what epidemic disease will attack the city, either in summer or in winter, and what each individual will be in danger of experiencing from the change in regimen. For knowing the changes of the seasons, the risings and settings of the stars, how each of them takes place, he will be able to know beforehand what sort of year is going to ensue. . . . And if it shall be thought that these things belong rather to meteorology, it will be admitted, on second thoughts, that astronomy contributes not a little, but a great deal, indeed to medicine. For with the seasons the digestive organs undergo change.

"Such waters," Hippocrates says, "as are marshy, stagnant, and belong to lakes, are necessarily hot in the summer, thick, and have a strong smell, since they have no current; but being constantly supplied by rain water, and the sun heating them, they necessarily want their proper color, and are unwholesome and form bile."

He speaks about the ill-health of the inhabitants of Phasis and says: "Their country is fenny, warm, and wooded. They drink the hot and stagnant waters, both when rendered putrid by the sun, and when swollen with rains. The Phasis itself is the most stagnant of all rivers, and runs the smoothest; all the fruits which spring there are unwholesome, of feeble and imperfect growth, owing to the redundance of water, and on this account they do not ripen, for much vapor from the waters overspreads the country."

Hippocrates, writing on "Acute Diseases," says: "Ardent fever (Causus) takes place when the veins, being dried up in the summer season, attract acrid and bilious humors to themselves; and strong fever seizes the body, which experiences aches of the bones and is in a state of lassitude and pain." The ardent fever that Hippocrates describes is the remittent fever of later writers. There are many references to it in Hippocrates' "Epidemics." In this latter work, he mentions that in Thasus, early during the summer and autumn, there were many dysenteric affections, attacks of tenesmus and lientery, bilious diarrhoea—vomiting of

phlegm and undigested food, sweats, in all cases a redundance of humors. "Persons died of all of these diseases," he says, "but mostly of these fevers, and especially infants just weaned, and older children until eight or ten years of age, and those before puberty. . . . Pains about the head and neck, and heaviness of same along with pain, occur either with or without fevers." He speaks about paralysis occurring in some of these individuals. In Thasus, following a hot summer and continued and severe drought, followed by southern winds, he found that an epidemic of paraplegia set in, and attacked many, and some died speedily.

"The mixture of salt and fresh water," Ludlow[91] (1823) states, "was early noticed by the ancients as producing more noxious exhalation than either uncombined. Sir John Pringle made experiments with a view of determining the cause, the result of which proved that salt in small quantities is a hastener of putrefaction."

Gardner[92] (1843) points out that sulphurated hydrogen gas exists in stagnant water, and the atmosphere of certain marshes and that this chemical has occupied the most prominent position to explain miasm. Gardner states that the intrusion of salt water into the marshes of Castra, near Venezuela, and at Sheerness, England make them exceedingly insalubrious.

In 1844, Channing[93], in Boston, was pleading, both in public and privately, for the introduction of pure water into the city of Boston. He pointed out that the water supply was from natural springs, was not pure, and hence unfit to use. In the same year (1844), Brande[94] stated that the water supplied to London was impure, excited disgust for it, and caused illness.

Story[95] (1848), reporting on Summer and Autumnal Fevers of the Connecticut River Valley, described an epidemic of fever in 1814, and another in 1825. Prominent symtoms included pains along the back of the neck and down the spine. "In June (1825), he says, "a severe drought commenced, which continued for nearly a year, so that wells, which had not known the want of water before, became dry, and some continued so for ten months." The epidemic that he describes commenced in July and lasted until November.

"The efficient cause of this epidemic appears to me to have been a malarious condition of the atmosphere, occasioned by the peculiarity of the season." He speaks of swampy grounds overrun

with rank vegetable growth, and which during the great drought of the season was in a decaying condition.

Bachelder[96] (1848) writing "On Epidemic Influence" points out that the water of a country or locality will influence the character of the complaints incident to such a place. "On the same principle food will have its effect," he says, "in determining the shape of disease. A person poisoned with a mixture of opium, aconite and belladonna, would manifest symptoms quite different from those which would arise from a destructive dose of either drug taken alone. So a person poisoned by human effluvia, marsh miasma and a vitiated atmosphere, acting in concert, will exhibit disorders essentially differing from those which would be the consequence of either of those morbific agents acting singly."

Bachelder points out that writers frequently allude to this, and hence, it has become a truism, that during epidemics, "the prevailing disease swallows up all other disorders." "Every indisposition of a febrile sort readily assumes the character of the prevailing disorder."

During the American Civil War (1861-1865), 2,907 soldiers were discharged from the Northern Army because of paralysis, and 272 deaths were attributed to this cause. Many of the outbreaks in the military camps that have been described appear to be identical with poliomyelitis, but they were designated by other names. A review of these epidemics has been reported by Scobey.[97]

The drinking water used by the soldiers was suggested as the cause of these illnesses that resulted in paralysis. Smart[98] accordingly instituted chemical studies of the drinking water and found that it contained a high percentage of nitrogen, which he attributed to the decay of vegetable and animal matter, and considered to be the cause of the disease.

Upham[99] (1863) described an epidemic of a cerebrospinal affection in an army camp in North Carolina. He points out that the weather was hot, humid, and subject to malarial influence in the summer and autumn. The water for culinary purposes was obtained mainly from wells sunk in the vicinity of the camp. It was brackish and unpalatable. River water was also, to some extent, used.

In its mode of attack, the disease was commonly sudden and without premonition. The subjects of the disease, in most cases,

were in the fullness of robust health—between the ages of 18 and 24 years—who had endured hardships and exposure with impunity.

The symptoms were at first headache, pain in the back and limbs, nausea and vomiting, chilliness, rather than a well-defined chill. Stiffness of the neck was often an early symptom. "In some cases the initiatory symptoms were those of a severe cold, with a disposition of paralysis of the tongue and a portion of the muscles of the face. With this the respiration would be difficult and irregular, giving occasion to a fear of a congestive attack of the lungs. There was often tenderness of the nape of the neck and along the spine early in the disease. . . . Stiffness of the muscles of the neck and back, or some perverted action of the muscles of the face was almost pathognomic," Upson says. He points out that there was considerable diversity in the manifestations of the disease, during its progress, whether toward a favorable or fatal result. The usual duration of the disease, he states, was from 3 to 4 up to seven days.

Smart[100] (1878) described conditions in the Wyoming Territory after his arrival with troops in August 1873. "The men complained that they had caught cold," he says, "and investigation showed the presence of slight catarrh or some equally slight congestion of the fauces; a local affection of too trivial a nature to account for the pecuilar coexisting constitutional disturbance. The bowels were constipated, urine scanty, skin dry, complexion sallow, pulse about 100, and temperature slightly increased, with loss of appetite and nausea, much languor and depression of spirits, pains in the bones and muscles, and stiffness of the joints. Occasionally instead of constipation there would be marked looseness of the bowels."

Smart made organic determinations of the drinking water at the station and the river water and found a high nitrogen content. He was satisfied that this was the cause of the illnesses; that it consisted of vegetable emanations and debris. "It has been a very general belief among the inhabitants of marshy countries," Smart says, "that the water could produce fevers."

During the epidemic of poliomyelitis which occurred in and around New York City in 1907,[101] and when physicians were looking for a specific germ as the causative factor, the lowness of the reservoirs due to lack of rain and the concentration of impurities

resulting from evaporation was being pointed out. During the same summer, a commission of Sanitary Engineers[102] showed that the Hudson River was polluted and constituted a menace. In New York City, there was an augmented mortality from malarial fevers and diarrheal diseases.

The Health Department of Brooklyn,[103] in 1908, issued a report to the effect that the water supply of Brooklyn was unfit for drinking purposes. Citizens of the Borough were advised against the use of the water except after it had been boiled. A chemist in the Health Department found that the water contained an excess of chlorides, in consequence of pollution of the reservoirs and infiltration from the seas.

Epidemics of progressive weakness, paralysis, and death in waterfowl and shorebirds, especially those in western lake regions, were found by Kalmback and Gunderson [104] (1934) to be caused by botulism. "The disease is caused by the toxic substances produced by the bacterium clostridium, type C, an organism that thrives best in an alkaline environment where there is an abundant supply of decaying matter and a reduced oxygen content. Subsidence of water, leaving pools little affected by flowage or wind action, is frequently followed by extensive outbreaks of this disease," Shillinger[105] says. The 'limber neck' of chickens is a well known disease, closely resembling human poliomyelitis and which has been observed during epidemics of this disease, also results from botulism. It results from eating feed or other material that has been contaminated. "Decomposed flesh and the maggots of flies which have bred on it are considered probable sources of botulinus poisoning," Bunyea[106] says. It may be possible, in the light of Kalmback and Gunderson's work, that it could also be caused by contaminated water.

"Fresh water plankton, especially blue-green algae, have been definitely linked to intoxications in domestic animals. Fitch and co-workers[107] (1934) have summarized the literature on this toxic 'waterbloom' and described cases of poisoning from it in fresh water lakes in Minnesota. The poison was also demonstrated in the filtered water."[108]

Lyon[108] (1942) makes the following significant remarks concerning this plankton poisoning: "The demonstration of this poison in the water of these lakes is of interest to those working

with the problem of stream pollution as it relates to drinking water. Some investigators have felt that the algae and other plant life which grow so profusely in our dammed up rivers may not only be responsible for changes in taste but even may be of importance in the production of acute gastro-enteritis when such water is drunk. On more than one occasion during periods of drought widespread occurrence of gastro-enteritis has appeared to be associated with water of poor potability, obtained from streams having a negligible rate of flow, and with heavy algae formation with the sluggish stream. It would not be surprising if there may not be derived therefrom some poisonous factor not totally unlike that responsible for 'waterbloom' poisoning. We have much to learn of marine toxicology and its relation to gastro-enteritis, no matter whether it be in salt or fresh water." Meier[109] (1939) points out that nitrogen in the form of nitrates is the principle factor that regulates the quantity of plankton. He states that algae readily multiply and its spores can be blown with the wind in dust and carried on the feet and feathers of birds from one body of water to another.

Tisdale[119] (1931), during an extensive drought in 1930-31, when one of the most severe epidemics of poliomyelitis recorded up to that time in this country was prevailing, noted that great numbers of fish were dying in the streams of Ohio, West Virginia, and Kentucky. The effects on the fish were noted when the taste of the public water supplies was bad and outbreaks of intestinal disorder were prevalent. There was an outbreak of so-called intestinal flu in Cincinnati and other river cities.

In the water in all of the affected states there was a prodigious algae growth in streams and storage basins with resulting nauseating tastes and odors. The theory was advanced at that time that the illnesses that were prevalent were due to a toxic property in river water by reason of decomposition of organic substances of vegetable or animal origin. Bacteriological examination showed that the water was safe to drink.

Ravenel[111] (1931), writing about the same drought and its effect on health, points out that there was an increase in the prevalance of intestinal troubles.

Schaut,[112, 113] Chief Chemist of Philadelphia Water Department, also noted that thousands of fish were dying in the Schuykill

River during this same period (1930-1931) and that gastro-intestinal disturbances among the people were prevalent. He observed that the fish showed intense activity, attempting to get out of the water, then became paralyzed and died. After exhaustive studies, including river surveys for factory wastes, and chemical examinations of the river water, river bottom mud, etc., Schaut concluded that the death of the fish was caused by 'waterbloom' which was present at the time of the death of the fish and had a smoky appearance and liberated a gas. He found on chemical examination that this water containing the 'waterbloom', also contained cyanide. He observed also that potassium sulfocyanate, a relatively non-toxic chemical, became very toxic for fish by chlorination. He noticed also, as a result of his experiments, that cyanide is produced in water when a very high chlorine demand is satisfied.

Schaut found that the water that he examined contained an enzyme that was capable of acting on cyanophore glucosides in the organic matter releasing cyanide. The enzyme was destroyed by boiling water but not by chlorination. He suggested that the detection of the specific enzymatic activity in water by chemical means may be an indication of on-coming epidemics. "More emphasis should be given the chemical and physical nature of water, rather than so much agitation about increasing the bacterial standards," he says. This mode of procedure of appraising the potability of water was also suggested for the investigation of epidemics by Hippocrates 2000 years ago.

Coinciding with the observations of organic chemical pollution of water by Smart,[98, 100] Kalmback and Gunderson,[104] Tisdale,[110] and Schaut, [112, 113] which have been mentioned, is that of Bertullo[114] of the Technological Laboratory, Fish and Wildlife Service, U. S. Department of Interior, College Park, Maryland. Bertullo found that as the water temperature in Chesapeake Bay increases, pseudomonas increase. He showed that these forms produce sufficient hydrocyanic acid to prevent the normal fermentation of lactose by the choleriform group. "This means that you have a water supply which would be approved on the basis of the bacteriological findings and at the same time be potentially very hazardous not only from hydrocyanic acid poisoning but also from intestinal pathogens."[114]

Increasing incidence of poliomyelitis epidemics during recent years parallels closely the incidence of droughts and water shortages. In California, where for several years the increasing incidence of this disease is alarming, there has been a critical water shortage due to lack of rain and the increasing needs for water. "Water for the rich farm lands of California and Arizona is getting scarcer and scarcer. Underground water is being used up faster that it is being replaced. Around Los Angeles many new wells already have lowered the fresh water table below sea level, so that salt water is seeping into wells on thousands of acres. Crops, particularly fruit, went largely unwatered." [115]

Oscar L. Chapman, Secretary of the Interior[116] (1950), makes the startling revelation in an article entitled, "Will there be enough Water for You?" that the water situation throughout the United States is serious. In a map that accompanies this article, areas are shown where critical water shortages exist. It is impressive to note that these areas correspond with those that have usually reported the greatest incidence of poliomyelitis. The outstanding example of a parallelism between epidemics of poliomyelitis and water shortage is that of New York City during the epidemics of this disease in 1907, 1916, and 1949.

PARALYSIS FROM FISH POISONING

The symptoms of poliomyelitis have not infrequently followed the eating of fish. Chisholm[117] (1808) states: "The many instances of the poison in fish, and the frequency of death in consequence of receiving it into the human stomach, do not seem to have raised to any useful degree, the attention of medical men. We cannot suppress our surprise that so little has been done towards obtaining a more perfect knowledge of the subject."

Chisholm described poisoning in three stout negro men following the ingestion of the muranea conger, common off the coast of Grenada. The symptoms in these cases continued for a fortnight and terminated in paralysis of the lower extremities. Fishermen pointed out to Chisholm that the flesh of the fish becomes poisonous to man during the months of May, June and July as a result of the food taken by the fish. At other times the same fish is wholesome and non-poisonous. "Some men," Chisholm says, "escape the effect of the poison of the fish at one time and

are affected by it at another." Galen, he points out, lays down the axiom that the most poisonous of substances, as hemlock, if introduced gradually into the body will not injure. Chisholm points out that lime and sugar cane juice, as well as sea water and alkalies, are effective in protecting against the poisoning by fish.

Chisholm observed that the poisonous barracuda is observed to have dark colored teeth and swelled bloody gums, as well as swollen and corroded chops; and that these fish seem to be in a languid state at the time, and emit a peculiar odor.

These observations correspond with those noted by modern fishermen in the United States. They point out that fish have a sore mouth condition in the month of August—during the dog-day season—and consequently will not bite. Such fish, many maintain, are unfit to eat. It is pointed out that the waters where these fish are caught are in "bloom", especially lakes that have grown stagnant from lack of fresh water coming in and old water being washed out.[118]

Lincoln[118] (1947) states: "When thick patches (of blue-green algae) appear on the water, often looking like the blue-green mold on the top of spoiled fruit in jars, it can be reasoned that the water is in fairly bad shape. Decomposition of vegetation and other contributing factors are sure to bring on the green scum mentioned." These facts correspond with the observations made by physicians of the past, including Hippocrates, who would obviously have designated such conditions in water as a source of miasm.

Burrows[119] (1815), England, in describing two cases of death from the eating of shellfish, states: "Many fish as salmon, herrings, eels, mackeral, many of the testaceous and most of the crustaceous fish of the country have occasioned symptoms similar to those of the poison of tropical fish. . . . The symptoms from eating land crabs, many drupaceus fruits, almonds, etc., are strikingly analogous to those of fish poison. The black land crab, which at certain seasons is deemed in the West Indies a great delicacy, at other seasons occasions the same symptoms as those arising from fish poison." This the author attributes to the feeding of the crab on the leaves and produce of the mountain mahault, or manchenelle. "As proof of this if crabs are placed in coops and fed wholesome vegetables they do not become poisonous," he says.

Burrows points out that fish eaten on the same day that they

are caught may produce no bad effects; but on the following day they may be poisonous. He says: "The flesh of fish is in all temperatures prone to a more rapid decay than that of warm blooded animals and it is certainly very probable, that if fish be caught in this unhealthy condition, it will, upon exposure to an intense degree of atmospheric heat, be liable to a sudden decomposition or putrefaction will, in a great degree, be regulated by its state of health. A sick fish, therefore, will exhibit its deleterious properties though eaten immediately it was caught; while a healthy one will be wholesome on the first day, prejudicial on the second, and on the third produce virulent effects." Burrows considered fish poison to be prussic acid, which is known to exist both in animals and vegetables. He points out that lemon juice, alkalies and sea water will prevent the poisonous effects of fish.

Lincoln[118] states: "I must remark that fishermen unwittingly permit thousands of fish to spoil during the hot weather period by leaving them on a stringer in the water off the side of the boat. . . . Remember, always, that decay of fish sets in at the gills and along the venous blood tract along the spine. . . . Fish die in so many minutes on a stringer in hot weather and almost directly start spoiling. It is highly possible that many have been sickened by eating half-spoiled fish; these fish may not smell strong, but still they will have 'turned.'"

A fact bearing on the preceding and the etiology of poliomyelitis is found in the affection of five dogs on the Labrador coast in the autumn of 1907.[120] The dogs were feeding on decomposed herring and were affected by a sudden paralysis, most marked in the hind legs. Three died and two recovered.

Mussels, clams and sand crabs may, during certain seasons of the year (summer and fall), and in certain years, contain a powerful neurotoxic poison which can produce paralysis or death from respiratory paralysis. Meyer and his co-workers[121] (1928) traced the poison to plankton upon which the mussels feed. The poison was demonstrated in the plankton residue and it was found that relatively large numbers of the dinoflagellate of Genus Gonyaulax contained demonstrable amounts of the neurotoxin.

In non-fatal cases of this paralytic shellfish disease, besides paralysis, gastro-enteritis with nausea, vomiting and diarhea are

to be found. Stegeman[122] (1934) found that the addition of baking soda to cooked mussels reduced or eliminated the poisonous effects in experimental animals.

Outbreaks of paralytic shellfish disease were first reported in 1885, in districts around the North Sea in Europe from which general region, and at about the same time, poliomyelitis was reported. The first extensive epidemic of this paralytic shellfish disease in the United States was observed in California in 1927. There were 102 cases reported with six deaths. In the same year this state reported its greatest number of cases of poliomyelitis (1,298 cases). The vast majority of cases of this disease were reported in the water-shed of the San Juan, Sacramento and other rivers, near the mouths of which the cases of paralytic shellfish disease were reported. These facts suggest a common etiological poisonous factor carried in the water responsible for both of these diseases.

Toomey et al.[123] (1945) experimented with fish caught in a polluted brook and in the mouths of two small rivers which emptied into Lake Erie, in the vicinity of Cleveland during an epidemic of poliomyelitis during 1941. Employing the gastro-intestinal content of several fish (mostly carp) which had been treated with ether to render it sterile, they were able to produce destruction of the anterior horn cells of the spinal cord of a monkey. In subsequent experiments, using the combined gastro-intestinal content from at least four dozen fish caught at the mouth of one of the rivers for their injection material, they were able to produce weakness in the legs of a monkey seven days after the inoculations. This animal was sacrificed and a suspension from its cord when injected into a second monkey caused weakness of both legs on the fifth day. An emulsion of the spinal cord of this monkey, when injected into a third monkey, caused weakness of the arms and paralysis of the left leg. Subtransfer of its cord to another animal was negative.

It appears not at all unlikely that the gastro-intestinal content of the fish examined by Toomey et al. contained poisonous plankton. This possibility is indicated by the fact that investigators studying paralytic shellfish disease have found the intestinal contents of poisonous mussels to be toxic for experimental animals.

PARALYSIS FROM MIASM

One has only to examine critically the literature of the past and it will be found that poliomyelitis beyond doubt existed, but that it was designated by many names. The writer has found individual cases and epidemics of paralytic diseases listed by nearly 200 names prior to 1890. Sometimes the same disease has been designated by different names in the same locality by different physicians. The writer[37] has already reported these facts in a previous paper. Table 1 includes those that have already been reported as well as additional names that have been found since that report. Some of the conditions that were included in the miasmic diseases of the past were without doubt the poliomyelitis of today.

TABLE 1. *Nomenclature of Poliomyelitis and Related Paralytic Diseases Prior to* 1890.

Acute anterior infantile paralysis
Acute anterior myelitis
Acute anterior paralysis
Acute anterior poliomyelitis
Acute anterior paralysis
Acute anterior spinal paralysis
Acute anterior spinal palsy of the adult
Acute anterior tephromyelitis
Acute ascending paralysis
Acute atrophic anterior poliomyelitis
Acute atrophic spinal paralysis
Acute cerebrospinal paralysis
Acute hydrocephalus
Acute infantile spinal paralysis
Acute inflammation of the anterior horn cells of the spinal cord
Acute meningitis
Acute myelitis
Acute myelitis of the adult
Acute myelitis of the anterior horns
Acute paralysis
Acute polioencephalitis
Acute spinal infantile paralysis
Acute spinal palsy
Acute spinal paralysis
Acute spinal paralysis of adults
Anterior poliomyelitis
Antero-spinal paralysis
Apoplexia hydrocephalica
Apoplexia infantum
Apoplexia serosa
Idiopathic meningitis
Idiopathic multiple neuritis
Idiopathic paralysis
Inertia of prostration
Infantile cerebral paralysis
Infantile fever
Infantile palsy
Infantile paralysis
Infantile paraplegia
Infantile remittent fever
Inflammation of the anterior columns of the spinal cord
Inflammation of the motor tract of the spinal cord
Inflammation of the spinal marrow
Inhibitory palsy
Inhibitory paralysis
Inter alia
Intermittent fever
Irritative fever
Irritative fever of children
Landry's paralysis
Low nervous fever
Mal'aria
Malaria
Malarial fever
Malarial neuritis
Medullary paralysis
Meningitis
Miasmic disease
Miasmic fever
Morning paralysis
Multiple neuritis
Myelitis
Myelitis of the anterior cornua

Apoplexy
Arachnitis
Ardent fever
Asthenic paralysis
Atrophic fatty paralysis
Atrophic infantile paralysis
Atrophic palsy of childhood
Atrophic paralysis
Atrophic paralysis of childhood
Atrophic spinal paralysis
Atrophy of anterior cells
Autumnal fever

Bilious fever
Bilious remittent fever
Bowel disease
Brain fever

Carus
Causus
Cephalitis
Cerebral infantile fever
Cerebrospinal arachnitis
Cerebrospinal meningitis
Cerebrospinal paralysis
Cholera
Cholera morbus
Chroea sancti viti
Chronic anterior poliomyelitis
Chronic atrophic spinal paralysis
Chronic meningitis
Chronic myelitis
Chronic rheumatism
Congestion
Contagious fever
Convulsive fever
Cornual myelitis
Cruveilhier's atrophy

Dental paralysis
Developmental fever
Disease of the season
Disease of the anterior cornua of the cord
Duchenne's paralysis

Endemic fever
Epidemic cholera
Epidemic fever
Epidemic poliomyelitis
Erb's juvenile progressive muscular atrophy
Ergotism
Ergot poisoning
Essential infantile paralysis
Essential paralysis
Essential paralysis of children

Myelitis of the anterior horns
Myogenic paralysis
Myopathic paralysis of children

Nervous apoplexy
Nervous fever

Organic infantile paralysis

Painful paralysis
Palsy
Palsy by atrophy of anterior cells
Palsy of motion
Paralysis
Paralysis of children
Paralysis of childhood
Paralysis of early life
Paralysis of exhaustion
Paralysis from foul bowels
Paralysis of infants
Paralysis of motion
Paralysis from peripheral irritation
Paralysis from teething
Paraplegia
Phrenitis
Phrenitis aestivale
Poliomyelitis
Poliomyelitis chronica
Progressive atrophic paralysis
Ptomaine poisoning

Raphania
Reflex paralysis
Regressive paralysis
Remittent fever
Resolutio nervorum
Rheumatic paralysis
Rheumaitc spinal paralysis
Rheumatic multiple neuritis
Rheumatism

Spasmo-paralysis
Spastic infantile paralysis
Spastic spinal paralysis
Spinal anterior paralysis
Spinal apoplexy
Spinal atrophic infantile paralysis
Spinal atrophy
Spinal infantile paralysis
Spinal irritation
Spinal paralysis
Spinal paralysis of the adult
Spinal spastic paralysis
Spinal anterior general paralysis of Duchenne
Spontaneous hydrophobia
Subacute general anterior spinal paralysis
Subacute posterior poliomyelitis anterior
Subacute spinal paralysis
Summer and autumn disease
Summer and fall fever
Summer complaint

Famine fever	Summer fever
Fever	Summer sickness
Food poisoning	Teething
Functional paralysis	Teething paralysis
General spinal paralysis	Transient paralysis
Hydrocephalus	Typhus fever
Hydrophobia spontaneous	Wasting palsy
Hydrocephaloid disease	Worm fever
	X-fever

John Maria Lancisi,[124] an Italian physician, was the first to put forth definite views on the subject of miasm as a cause of disease. In a work entitled, "De Noxiis Effluviis" printed in 1717, he advanced the idea that intermittent and remittent fevers were caused by certain marshy exhalations, to which he gave the name "miasmata," from the Greek word "to pollute." Although at the present time we consider the word "malaria" to indicate a distinct disease entity, it was often employed in the past as the substance responsible for a group of diseases of which the modern disease, malaria, was only one. The word malaria (mal'aria, marsh miasm) means bad air, and as such was denominated as the cause of disease in the past rather than the name of a distinct disease entity. Thus we find references to many illnesses of the past, some resembling our modern poliomyelitis, designated malarial disease, malarial fever, miasmic disease, etc. Boyle[125] (1810) says: "Among the vulgar, both in Sicily and Calabria, most of the diseases peculiar to the climate, particularly those of a malignant nature, are attributed to what they call mal'aria."

Schoeppf[126] (1788), who was a surgeon of the Auspach-Bayreuth troops of America during the Revolutionary War, devoted two years to travel throughout the middle and southern United States, and wrote on the diseases of America that were prevalent at that time. He speaks about a great deal of sickness during those two years. "There is a belief, founded upon universal experience," he says, "that the variety of fever of which we are speaking is chiefly generated by emanations from foul, marshy, and stagnant water; at all events, they abound chiefly in districts of this description. . . . The surface of the body is so sensitive to the slightest influence, when the heat is very great, that nothing is commoner than to meet people with catarrhs and colds at such times."

(To be concluded in October issue)

PEDIATRICS HALF A CENTURY AGO

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

ACID AUTOINTOXICATION IN INFANCY AND CHILDHOOD*

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Vicarelli (1895) examined the urine of thirty perfectly healthy infants under six months for acetone. The distillate of the twenty-four hour amount was almost always positive by the iodoform test, but negative with sodium nitroprussid and oxalate of mercury. He concluded that the urine of healthy sucklings does not contain acetone. Baginsky (1888) found that acetone was present in the urine of healthy children under apparently entirely normal conditions, but only in very small quantities; while Langstein and Meyer (1905) found that the normal excretion in twenty-four hours never exceeded 1 Cgm. Schrack (1889) says that the urine of children often contains small amounts of acetone from very insignificant causes. Brackett, Stone and Low, however, found acetone in only eleven of 300 surgical cases at the Boston Children's Hospital. In them it was associated with more or less severe symptoms. Langstein and Meyer (1905) found that when children from six to fourteen years of age received a meat diet without carbohydrates relatively more acetone was excreted than in adults. They also found that the principal part of the acetone was excreted through the lungs, and not through the urine as in adults. They always found diacetic and B-oxybutyric acid in considerable amounts. The organism of the child invariably reacted to the acidosis in the body by an increase in the excretion of ammonia. Camerer (1903) states that the nitrogen of ammonia in infancy is unexpectedly high.

* Read before the Seventeenth Annual Meeting of the American Pediatric Society held at Lake George, N. Y., June 19, 1905.
Reprinted from ARCHIVES OF PEDIATRICS, 22:561-571, Aug. 1905.

The work of von Jaksch (1886) on acetonuria and diaceturia inspired Baginsky (1888) to study acetonuria in children. Schrack (1889) in the next year examined the urine of 400 children for acetone and diacetic acid. Their results and those of others, Bottazzi (1901), Freund (1903), Bernert (1904), Meyer (1904), in smaller series of cases, including one of 150 of my own, tested for both acetone and diacetic acid by the sodium nitroprussid and ferric chlorid tests, apparently show that the acid bodies are present in the urine of infants and children under approximately the same conditions as in adults. They are often present in acute diseases accompanied by high fever (especially in the acute exanthemata, diphtheria and pneumonia), vary, as a rule, directly with the height of the fever and disappear with the fall of the temperature. I found but two positive reactions in 20 cases of pneumonia. In both the reactions occurred at the height of the fever, while the patient was taking sufficient food; and in both the reactions disappeared immediately after the fall of the temperature. In 7 negative cases the food was markedly insufficient in amount, although it contained a fair proportion of carbohydrates. These facts seem to show that the acid bodies were directly dependent upon the temperature. Meyer, however, claims, as the result of his observations in diphtheria, scarlet fever and measles, that acetonuria in the infectious diseases is independent of the sort or intensity of the infection and of the degree of fever, being due solely to carbohydrate inanition which is favored by the diminished power of carbohydrate metabolism in these diseases. In some instances, especially in typhoid, the acid bodies may persist, or first appear, during convalescence. They are then probably due to insufficient food or carbohydrates. In 15 cases of typhoid, in my series, 23 negative examinations were obtained in 13. The temperature was high in all these cases, and the food insufficient in amount in 4. Positive reactions were obtained in 2 cases at the beginning of convalescence; in one of these it persisted for eight days and only ceased when a sufficient amount of food was taken, although the food before was milk and barley-water. Acetone was present in 1 of 3 cases of bronchitis, and diacetic acid in 1 of 2 cases of pleurisy with effusion, while both were absent in 1 of empyema. Neither acetone nor diacetic acid were found in 2 cases of malaria, 3 of cerebrospinal meningitis, 2 of otitis media,

2 of pyelonephritis, 2 of rheumatism, 2 of cystitis and 1 each of acute nephritis and mastoid abscess. The food was markedly insufficient in several of these, although all took some milk. The acid bodies are said to be seldom present in tuberculosis of the lungs and intestine, but almost always in tubercular meningitis and miliary tuberculosis. They were absent, however, in 3 cases of tubercular meningitis, all of which were taking almost no food, and in 1 of tuberculosis of the kidneys in my series.

The acid bodies in the urine are enormously increased in children during convulsions. Löbl found all the acetone bodies in a series of cases of tetany even when gastrointestinal symptoms were not marked. I did not find them in a case of tetany or in 1 of mild tetanus. They are, however, probably not the direct cause of the convulsions, because they are absent or present in only small traces in the urine of those diseases in which convulsions are most common, and because they may be present in large amounts without producing convulsions. They are not due to the lack of oxygen during convulsions, because lack of oxygen in conditions without fever does not cause them. The acid bodies and the convulsions are probably, therefore, independent of each other and merely manifestations of some common cause.

While the effect of fasting in the production of the acetone bodies has been pretty carefully worked out in adults, there are few or no data on the subject in infancy and but few in childhood. The effect of variations in diet has been but little studied at this age. Schrack (1889) never found the acetone bodies while a pure milk diet was being taken. They were found in a certain proportion of cases on a pure meat diet, and disappeared when an exclusively carbohydrate diet was substituted. Langstein and Meyer (1904) found the acetone bodies earlier and in relatively greater quantities in the urine of children from six to fourteen years of age who were fed on meat without carbohydrates than in adults under similar conditions. They assume that this is because the carbohydrate reserve in the liver and muscels is smaller than in adults. They call attention to the great danger of a fatty diet in children, when there is any disturbance of the carbohydrate metabolism. Czerny's and Keller's (1897) experiments on the excretion of ammonia in infants with gastrointestinal disorders point in the same direction. They found that the excretion of

ammonia was much increased when creams low in proteids and sugar were given, while it was not increased when milks high in proteids and low in sugar or diluted skim milk with sugar added were given.

I have had no opportunity to study the effect of starvation for periods of more than forty-eight hours in infants and children. The food was withdrawn in all these cases purely for therapeutic reasons and not for experimental purposes. Neither acetone nor diacetic acid were found in the urine of infants under two years under these conditions, but diacetic acid was found in a child of two, and acetone in a boy of nine years, after twenty-four hours of starvation. It quickly disappeared in both when milk was given. No acetone bodies were found in fifteen infants that had taken markedly insufficient amounts of food for considerable periods and were not gaining or were losing in weight. The food, being some form of modified milk, always contained, however, relatively large amounts of carbohydrates. What little evidence there is goes to show, therefore, that the effects of starvation and variations in diet are essentially the same, although more marked, in childhood as in adult life. The data for infancy are too limited to warrant any definite conclusions, although they seem to suggest that the acetone bodies do not appear in the urine as the result of starvation as soon as in later life.

Both acetonuria and diaceturia had been previously noted in the acute gastrointestinal diseases of infancy by von Jaksch, Schrack and Baginsky. Attention was more particularly called to acid intoxication in these diseases, in 1897, by Czerny, Keller and Van den Bergh. Keller first noted that the excretion of ammonia was increased both relatively and absolutely. This has since been confirmed by Camerer (1903). Van den Bergh showed that this increase was due to an increase in the acids in the blood because it was diminished by the administration of alkalies. Czerny attributed the acid intoxication to abnormal products of decomposition of fats and proteids, but did not mention the acetone bodies specifically. The work and conclusions of Czerny and his school have been considerably discredited, however, in recent years. Vergely, in the same year, reported 20 cases in children, 5 of them in detail, in which he found the acetone bodies. In 1 case the course resembled that of typhoid fever. The acetone bodies were

found as soon as they were sought, sometimes within a few hours after the onset. The acetone varied in amount from 10.5 to 12.5 grams per litre, and the B-oxybutyric acid from 6.8 to 8.5 grams per litre. The cessation of the symptoms and the disappearnce of the acetone bodies from the urine were simultaneous. He divides the symptoms in these cases into two classes, nervous and gastric. The nervous symptoms are change of disposition, excitability, restlessness, irritability and insomnia; the gastric, anorexia, vomiting, thirst and constipation. There was no enlargement of the spleen, but slight enlargement of the liver. The temperature was sometimes elevated, sometimes not. It never lasted more than ten days. He concludes that these digestive troubles have a peculiar symptomatology, which is due to the presence of these acid bodies. He believes that the reason that the symptoms of acid intoxication are not more severe is because the eliminative organs are very active in childhood. Litten (1883) described 3 cases of a peculiar symptom complex in young children. A prodromal stage of a few days of mild symptoms of disturbed digestion was followed by marked nervous symptoms. The children became apathetic, whined continually, and could not be induced to move, play or take food. Sleep was interrupted and accompanied by groans and cries. An aromatic odor then appeared in the breath and drowsiness took the place of sleeplessness. One of the children had slight convulsive twitching. The type of respiration was not changed. With the appearance of the drowsiness diacetic acid was found in the urine. After two or three days the odor in the breath, the diacetic acid and the drowsiness disappeared together, recovery then being complete. These symptoms were altogether out of proportion to the severity of those in the digestive tract. He considers, however, that the primary cause is a disturbance of digestion. In a somewhat similar case reported by Krotkov, the diet for several days before had consisted almost entirely of fat pork. Similar cases in adults have been reported by Litten, Lorenz (1891) and Edsall (1902). Czerny (1897) has called attention to cardiac weakness and slowing of the respiration in infancy as the result of toxemia in the gastrointestinal canal, and says that the symptoms are like those of acid intoxication in animals. He does not mention the acetone bodies specifically.

All observers agree that the acetone bodies are rarely found

in the urine in the chronic gastrointestinal disorders of infancy. My own experience leads me to believe that they are almost never present either in acute or chronic gastrointestinal diseases at this age. It is rather surprising that they are not because the limits of assimilation for sugar in infants with gastrointestinal diseases are much diminished, and the conditions for the development of an intermediary acidosis are thus present. In a number of these cases, however, I have found sugar in the urine, but no acetone bodies.

The conditions seem to be markedly different in children, however. At this age the odor of acetone is not infrequently present in the breath, and the acetone bodies in the urine, not only in acute, but also in chronic disorders of the gastrointestinal tract. The odor of acetone in the breath, and the presence of acetone and diacetic acid in the urine, are especially common in the type of indigestion known as "chronic duodenal" not only during exacerbations but also in the intervals. Lorenz (1891) has already called attention to this fact in adults. I have found them pretty constantly in 2 cases during one and two years, respectively. This is not surprising, however, since the digestion of fat is especially interfered with in this condition. In a number of other children under my care, who are subject to disturbances of digestion, the odor of acetone in the breath is among the earliest, and is often the earliest, indication of an impending attack of indigestion. Examination of the urine at this time sometimes shows the acetone bodies, sometimes not. The peculiar odor in the breath is often associated with marked restlessness and irritability for some days before the development of other symptoms. Apparently impending attacks of indigestion can often be aborted or prevented in these cases by the use of bicarbonate of soda. In these cases the acid bodies appear when food sufficient, not only in quantity, but in the relative proportion of carbohydrates is being taken, and before the appearance of acute symptoms. They cannot, therefore, be attributed to starvation. These cases, while they resemble those in the group to be described later as recurrent vomiting, are more nearly like those described by Vergely. The symptoms of nervous irritability are marked and out of proportion to those of indigestion. They are sometimes accompanied by various forms of urticaria. The nervous symptoms cease with the disappearance of the acetone bodies.

There is no doubt that the acetone bodies are often present in small amounts in the urine in both acute and chronic gastrointestinal disorders in which there are no symptoms whatever of acid intoxication. In these cases they are certainly of no importance. There is no question, moreover, that in other cases there are marked peculiarities in the symptoms, especially in those referable to the nervous system, which coincide with the appearance and disappearance of the acetone bodies. In these cases we are certainly justified clinically, in spite of the small amount of these bodies, in concluding that they play a certain part in the production of the symptoms. We are not justified, however, in concluding that acid intoxication is the only cause of the symptoms. In fact, it almost certainly is not. In certain cases, nevertheless, in which the symptoms resemble more closely those of diabetic coma, it probably is the most important cause.

There is great difference of opinion as to whether the acetone bodies are formed in the intestine in these cases, or in the body as the result of abnormal intracellular processes. The weight of evidence seems to show that they are not formed in the intestinal canal. It is probable, however, that they are indirectly dependent upon intestinal derangements which produce complex nutritional disturbances, which, in turn, result in the formation of the acetone bodies. The primary cause is almost certainly located in the intestine. Although the acetone bodies are secondary products, they do, in many cases, cause more or less characteristic symptoms, which not very infrequently dominate the symptom-complex. The demonstration of their presence is unquestionably, therefore, of value, both in diagnosis and in treatment.

Under the name of "vomiting in acetonemia," Marfan (1901) described a series of cases in children apparently identical in their symptomatology with those described in this country as "cyclic," "recurrent," or "persistent vomiting." The odor of acetone in the breath was always present at the beginning of the vomiting, and in 2 cases he detected it before the onset of the vomiting, showing that it was not due to inanition secondary to the vomiting. The odor of acetone either disappeared with the cessation of the vomiting or persisted for a day or two longer. Acetone was always, and diacetic acid sometimes, present in the urine. The amount of acetone in three cases was 0.38, 0.40 and 0.60 grams

per litre, respectively. In some cases the acetonuria persisted for weeks after the cessation of the vomiting, although the children seemed in perfect health. In two instances several children in the same family were attacked simultaneously. He thinks that mild symptoms, such as loss of appetite and lassitude, may for a short time precede the onset of the vomiting. He thinks that these attacks have no connection with the kind of food, and that the exact cause is entirely unknown. He admits that the acetonemia, by itself, has no precise significance, and considers it merely as an indication of a disturbance of the nutrition which may develop under various circumstances. He believes that the presence of acetonuria at the onset shows that it is not due to inanition, and that the disease does not originate in the stomach. He is not justified, of course, in assuming the presence of acetonemia because of the presence of acetone in the urine and breath. If the term, "vomiting in association with the presence of the acetone bodies," is substituted for the term used by him, his argument seems sound, however. In the discussion of a somewhat similar case reported by Guinon (1902), Guinon, Merklen and Siderey all stated that in their opinions regulation of the diet and attention to the bowels will not prevent the occurrence of these attacks. Barbier also stated that in his experience the odor of acetone always preceded the onset of the attacks. In one of his cases the urine contained about ten times the normal amount of acetone per kilo. Edsall, in the next year (1903), reported several similar cases very briefly, including some of the cases reported more fully a little later by Peirson. In the case most carefully observed by Peirson, acetonuria and the odor of acetone in the breath always preceded the appearance of the vomiting; in 2 others, acetone and diacetic acid were found early in the attacks. He thinks that the onset of the vomiting is always preceded by other mild premonitory symptoms, as well as by the presence of acetone in the breath and urine. These symptoms are slight rise in temperature, loss of appetite, abnormal movements or slight nervous manifestations. He thinks that after these symptoms have appeared change in the diet will not prevent the attack. Both Edsall and Peirson succeeded in aborting or relieving the attacks by the free use of bicarbonate of soda.

In addition to seeing 2 of Peirson's cases, I have seen 2

others in which the odor of acetone in the breath, or acetonuria, preceded for twenty-four or forty-eight hours the onset of the vomiting. In several others I have found acetone or diacetic acid within the first twenty-four hours after the onset. In my cases also careful inquiry always revealed a prodromal stage of a day or two of slight indigestion, or nervous symptoms. Bicarbonate of soda relieved or aborted the attacks. Diet neither prevented nor relieved them. Some of these cases resemble, to a certain extent, those described by Vergely and others under the head of acid intoxication in connection with gastroenteric diseases, the chief difference being in the mildness of the digestive symptoms before the onset of the vomiting. In others, however, it is very difficult to trace any connection with previous digestive disturbances or errors in diet. Moreover, regulation of the diet does not prevent their recurrence. In all, moreover, the severity of the symptoms is entirely out of proportion to the evidences of digestive disturbance. The presence of the signs of acid intoxication before the onset of the vomiting, their association with the vomiting, and the simultaneous subsidence of the two, certainly seem to show that they are intimately connected. The fact that the attacks of vomiting can be prevented, aborted or relieved by the administration of soda, the antidote for acid intoxication, shows that they must be due, in part, at least, to the acid intoxication. It is probable that the origin of the acid intoxication is not the same in all cases. In some it is probably due primarily to disturbances of metabolism, whose nature and cause are unknown, while in others it is probably primarily due to disturbances of digestion, which presumably induce secondary disturbances of metabolism.

It is undoubtedly true that the clinical picture of recurrent vomiting may be present without any evidence of acid intoxication and entirely independent of it. This fact does not prove, however, that in those cases in which the evidences of acid intoxication are present, it is not the cause, or one of the causes, of the attack. It merely shows that more than one pathological condition can produce the symptom-complex of recurrent vomiting.

It must be remembered, also, that the association of the evidences of acid intoxication and vomiting does not justify the diagnosis of recurrent vomiting to the exclusion of other conditions. This is well shown by a case reported by Leignen and Mirallie

(1904), in which after a few days symptoms of tubercular meningitis developed, and were followed by death in three weeks.

Brackett, Stone and Low (1904) recently reported a series of 13 very curious cases occurring at the Boston Children's Hospital, on the surgical side, both before and after operation. They also quote a series of similar cases from literature. These cases all showed certain symptoms in common. These were: "Vomiting, associated with collapse; a very weak and rapid pulse; absence of fever; cyanosis, in the fatal cases, causing extreme dyspnea, apathy and stupor, alternating with periods of restlessnes at first, but in the fatal cases, gradually deepening into coma and death; and the presence of acetone in the breath and urine." All these cases had extensive muscular atrophy as the result of paralysis or mechanical treatment. Autopsies were obtained in 4 cases. The only marked anatomical lesion made out was extreme fatty degeneration of the liver and muscles. They determined pretty conclusively that no single cause such as anesthesia, surgical shock, or change in diet, could have produced these symptoms. They conclude, however, that any one of these and other similar conditions may lead to changes in metabolism which, unless all the organs are sound and performing their functions normally, may produce serious or fatal complications. They attribute the symptoms to acid intoxication, and consider that the source of the acid bodies is the fatty degeneration of the muscles and liver. They finally conclude that the appearance of acetone in the urine, in quantities sufficient to give an ordinary clinical reaction, is to be regarded as an indication of serious, and possibly dangerous, disturbance of metabolism. Joslin estimated quantitatively the acetone excretion for twenty-four hours in 1 of these cases, a child of eight. The total excretion was 142 mgm., considerably more than the nuormal, but very much less than that found in diabetics, in whom it often reaches 100,000 mgm. This compartively small amount of acetone would seem to cast some doubt on acid intoxication, as the cause of the symptoms. In spite of this, however, the writer, who has seen a number of the cases, cannot help feeling that there is an intimate relation between the evidences of acid intoxication and the other symptoms in these cases, and that acid intoxication, whatever its cause, is the cause of the symptoms.

Conclusions. The acetone bodies are not found in the urine of comparatively healthy infants and children by the ordinary clinical tests. They appear in their urine under approximately the same conditions as in adults. Certain disturbances of digestion associated with the presence of the acetone bodies in early life have peculiar symptomatologies. It is probable that the peculiar symptoms are due, in part at least, to acid intoxication. It is also probable that the acid intoxication is not primary but secondary. The connection of the symptom-complex seen in many cases of recurrent vomiting with acid intoxication is probably even closer than in the digestive disturbances just mentioned. In these cases, also, the acid intoxication is presumably always secondary to some other abnormal condition, which may or may not be digestive in origin. In any event, the etiology is obscure. In spite of the fact that the amount of the acetone bodies found in these conditions is relatively much smaller than those found in diabetes, the demonstration of their presence in connection with symptoms of gastrointestinal disturbances and the symptom-complex of recurrent vomiting, and probably also with other conditions, is of importance both in diagnosis and in treatment.

ALLEGED ACCIDENTAL SUFFOCATION IN BABIES. (Medical Journal of Australia, 1: 65, Jan. 21, 1950). Bowden discusses the possible causes of death in infants who are found face downward or beneath the bed clothes, whose sudden death is attributed to accidental suffocation. Inspection of the baby in his bed just after death may show considerable cyanosis, especially of the ears, face and lips, further suggesting that death has occurred from suffocation. Careful inquiry shows that many of these children were not in perfect health before death. Past statistics on accidental suffocation have erred on the side of gross exaggeration in Australia and in the United States. A thorough microscopic investigation, including organs the appearance of which seems to the naked eye to be normal, will rarely reveal accidental suffocation. The author lists data on 40 babies who died suddenly. Most cases are explained by the fact that such disease as otitis media, upper respiratory tract infection and meningitis may overwhelm an apparently well infant.—*Journal A.M.A.*

CLINICAL-PATHOLOGICAL CONFERENCE

WILLARD PARKER HOSPITAL, NEW YORK

Meeting held June 20, 1950

DR. PHILIP MOEN STIMSON, presiding.

MEASLES ENCEPHALITIS

DR. HOFFMAN presented Case No. 2523, a 7-year-old white female, who was admitted to Willard Parker Hospital on April 15, 1950 with a history of measles since April 12, 1950. Patient's history further revealed an admission to Bronx Hospital, at the age of 5 years, after a car accident, with a concussion. No previous contagious disease. Normal growth and development. Has had all immunizations and vaccination as a baby.

Present Illness. There was an outbreak of a rash and high temperature 3 days before admission. On the day before admission, temperature returned to normal and the child appeared well. On the day of admission, her temperature spiked to 104° F. in the morning and she had 4 convulsions, lasting approximately 5 minutes each and described as bilateral and generalized by parents, occurring between the hours of 2-5 P. M. At this point the family physician was called and he administered 5 grains of sodium luminal by hyperdermic, 15 grains of aspirin by mouth, and called for a Willard Parker Hospital ambulance. One hour later, when the Willard Parker Hospital physician arrived with the ambulance, the temperature was still 104° F. The patient was comatose and did not respond to painful stimuli.

Physical Examination. Skin: Generalized fading maculopapular rash, partly hemorrhagic in type. Mucous membranes: Showed no Koplik's. Moderate degree of cyanosis. Eyes: Pupils were dilated and reacted sluggishly to light. Pharynx and tonsils were slightly enlarged and injected. Chest: Multiple medium moist râles throughout. No dullness. The lymph nodes were palpable and moderately enlarged in all areas. Neuro: Neck was moderately stiff to flexion. Kernig, Brudzinski and Babinski reflexes were positive. The abdominal reflexes were absent. Deep tendon reflexes: They were present in the upper but could not be elicited in the lower extremities.

Course. On arrival at Willard Parker Hospital her temperature had dropped to 100.4° F. She received on admission 10 cc. of globulin. A spinal tap was done, and clear fluid was obtained with no cells, protein 44 and sugar 130. A blood culture was taken which proved to be sterile. She was put on a regime of 200,000 units of aqueous penicillin every 3 hours, placed in an oxygen tent and suctioned frequently, a 5 per cent glucose solution infusion begun, and, consequently, her color improved somewhat and she responded to stimuli. According to nurse's notes, twitching of right hand, fingers and right side of face involving the right eye and right corner of the mouth occurred between the hours of 2-5 A. M. Around 6 A. M. the respiration became more shallow and increasingly rapid. Large amounts of pink mucoid material were suctioned from pharynx. The cyanosis became more marked as respiration became labored and irregular. The administration of caffeine and coramine in combination with artificial respiration was of no avail and the child expired 12 hours after admission. A total of 1,200 cc. of the glucose infusion had been given. Spinal fluid culture: Sterile. Postmortem nose and throat culture showed hemolytic staph. aureus.

Microscopic sections in this case shown by Dr. Dolgopol.

Pathological findings in this case showed perivascular cuffing was present in the gray matter of the cortex, in the basal ganglion, medulla and spinal cord. Extra-adventitial collections of glial cells were present around some veins of the gray and white matter, with resulting perivascular demyelination in the latter. There also was some marginal gliosis in the spinal cord. Some degeneration of myelin was observed in the optic nerves and the ganglion cells in all parts of the central nervous system showed marked chromatolysis.

MEASLES ENCEPHALITIS

Dr. Larsen presented Case No. 3269, a 4-year-old white girl, who had been well until 5 weeks before her admission to this hospital on May 17, 1950. At that time she and a younger sister developed an illness which was diagnosed by the local doctor as pneumonia. She was treated at home with penicillin and seemed to require "quite a few shots" and took much longer to get well than her younger sister. After about 2½ weeks, the child seemed

recovered from this illness, only to develop measles, 1½ weeks later.

Present Illness. She had the usual prodromal symptoms of fever, coryza and cough, followed by the typical measles rash which began 5 days before the night of admission.

After the rash was fully developed, the child seemed to improve, was alert, happy, would eat and had less fever.

On the day before admission, 4 days after the rash began, she became irritable, refused food, complained of headache, and began to urinate involuntarily. At about noon time, on the day of admission, 5 days after the rash developed, she began to fail to recognize her parents. According to their observation, she wanted to "sleep all the time," and her head began to "twitch." She vomited on one occasion that morning.

Physical Examination on admission to the hospital revealed a 4-year-old white girl, well developed, moderately nourished, lying quietly in bed, moaning at times with a dull, listless expression when not being handled. She was hyperirritable, would cry when approached, would answer when spoken to and called for her mother at different times. Temperature 100° F., pulse 96. Skin: Generalized, fading, maculopapular rash, typical of measles. Eyes: Reacted to light, but would not follow objects; roving. Ears: Drums normal. Throat: Moderately injected with a few fading Koplik's spots. Chest: Clear. Heart: Normal. Extremities: Good mobility, no paralysis. Reflexes: Knee jerks greatly increased bilaterally. Brudzinski, negative. Babinski, negative. Neck, supple. Abdominals, hyperactive. Spinal tap, done on admission, reported by Dr. Applebaum's Laboratory showed: clear fluid, slightly increased pressure, 70 cells, all monocytes, sugar 58 mg. % and protein 45 mg. %. No bacteria either by smear or culture. Immune globulin, 10 cc., was given at once and to be continued daily.

Course in Hospital: May 18, 1950. Condition seemed more serious the following afternoon. At that time, the child was unable to swallow fluids, and 500 cc. clysis of normal saline and 5 per cent glucose was started followed by 500 cc. 5 per cent glucose in distilled water. Temperature remained near 100° F. but pulse rose to 120. May 19, 1950, the second day after admission, the child continued to be sustained by parenteral fluids. She began

to show athetoid movements of her hands, irritability to being handled but would no longer respond to the spoken voice. She became more stuporous, would not respond to painful stimuli, and in the afternoon her extremities became spastic. Temperature 99° F. Pulse remained near 120. She was placed in an oxygen tent and given 300,000 units procaine penicillin at this time. May 20, 1950, 1:00 A. M., the child had a generalized convulsion with twitching of the entire body, chewing of the tongue, complete rigidity of extremities and a moderate amount of mucus. The convulsion lasted 2½-3 minutes. After this there was only partial muscular relaxation and child remained in coma. At 2:00 A. M. the child had 3 more convulsions of a similar nature. Her pulse grew weak and thready, and her respirations became shallow. The child continued in complete coma, and expired 4:40 A. M. despite intracardiac coramine and caffeine. Death occurred 2½ days after admission and 8 days after the beginning of the rash.

Laboratory Examination revealed the following:

Complete blood count on May 19, 1950: W.B.C. 12,550 per cent, polys. 81 per cent (few toxic granules), lymphs. 17 per cent, large monos. 1 per cent, basophiles, 1 per cent, R.B.C. 4,430,000, hemoglobin 14.5 gms., 96 per cent.

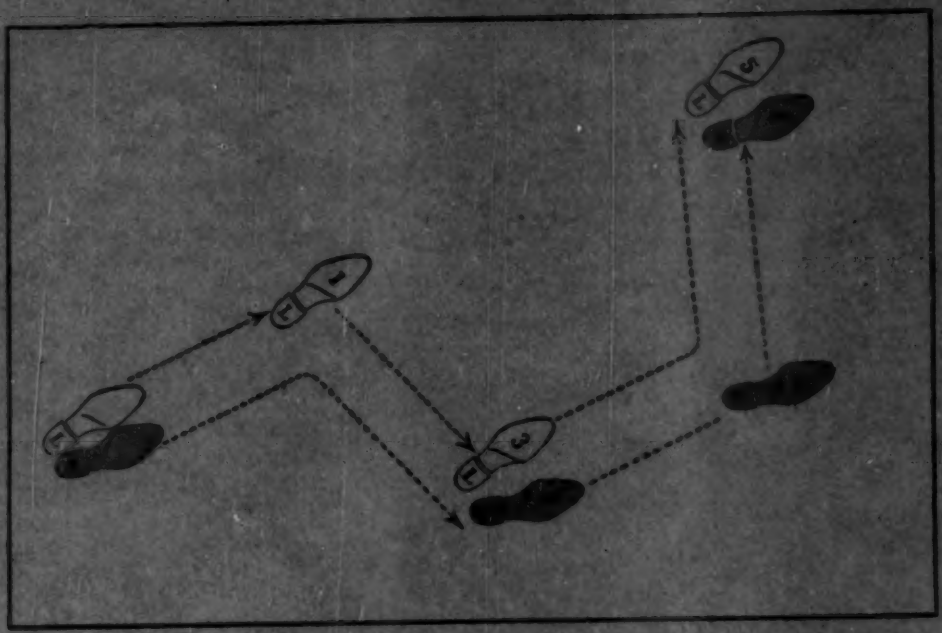
Postmortem Laboratory Studies. Spinal tap, May 20, 1950. Clear fluid. Globulin—trace. No cells. Sugar 143 mg. per cent. Icteric index 9.8. Van den Bergh—negative to both direct and indirect. Cephalin flocculation—2 plus. Blood culture—sterile. Nose and throat culture—staph. aureus hemolyticus.

Sections of the brain of this 4-year-old child showed a number of adventitial mononuclear infiltrates around the veins of various parts of the gray and white matter of the cerebral cortex, in the medulla, in the gray matter of the lumbar and sacral cord and a few minimal perivascular cuffs in the thoracic and cervical gray matter. Marked increase in glial elements was found in the cortex and some small glial collections were found in the white matter of the cortex near cuffed blood vessels. The myelin showed no significant damage, but, of course, the glial collections had pushed aside the myelonated fibers. The ganglion cells were essentially intact in the brain, cord and medulla, except for some cellular damage in the reticular substance.

ITEM

Encephalopathy After Combined Diphtheria-Pertussis Inoculation. (Lancet, London, 1: 537, March 25, 1950). Anderson and Morris report on a boy (one of uniovular twins) who had normal health and development until the age of 2 years and 4 months, when he was admitted to the hospital because of a generalized convulsion. The duration was short, left with transient right hemiplegia for five hours. He was discharged from the hospital the next day. At the time of this illness three other children in the same house were suffering from mumps, and his illness was diagnosed as mumps encephalitis. At the age of 2 years and 7 months, the twins were immunized with combined diphtheria-pertussis antigens. One twin remained well, but the patient had a generalized convulsion 36 hours later. Investigations made eight months later showed dilatation of the left lateral ventricle and diminished electrical activity of the left cerebral hemisphere. A year later there was some recovery, but the patient still had a small vocabulary and was mentally retarded. The pathogenesis is unknown, but, in view of the reports of serious neurologic complications, it is suggested that pertussis vaccination should not be done if there is a history of convulsions in the child or the family.—*Journal A.M.A.*

Hereditary Cataract. (Acta Ophthalmologica, Copenhagen, *27*: 537, 1949). Warner reports a Danish family in which cataract has been traced through eight generations. The family was first mentioned in medical records in 1878, when 20 of the 25 members were found to have cataract. In 1896 there were 40 with cataract among 69. Today the family consists of 542 members, of whom 132 have cataract. The hereditary transmission is dominant, since half the offspring of those with a cataract have the eye defect. The incidence is about the same in both sexes. The cataract is bilateral in all cases, and in most it is the same in both eyes. It is a characteristic feature of this family that, in contrast with most cases of congenital cataract, this cataract is not stationary but increases in density more or less rapidly, a circumstance that is documented in 11 cases by repeated examination.—*Journal A.M.A.*



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It will take approximately two minutes to find out how you can invest in either one of the two automatic plans offered by U. S. Savings Bonds.

Inquire at your place of business about the Payroll Savings Plan. Learn how easy it is to put extra savings for Bonds right from your paycheck.

Or, if you have a checking account, ask at your bank. They'll explain all about the Bond-A-Month Plan, in which you use your checking account as a means for investing in U. S. Savings Bonds.

Both plans are simple, and call for no effort on your part.

Both plans add up to the same thing—money to live on in the future.

So start finding out about them. Remember, it only takes two minutes today to learn how you can make your tomorrows a lot more carefree!

Automatic saving is *sure* saving—U.S. Savings Bonds

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They've heard the call for

VI-DAYLIN

TRADE MARK

(Homogenized Mixture of Vitamins A, D, B_1, B_2, C and Nicotinamide, Abbott)

MAYBE it doesn't happen quite like this, but children do "fly for home" when VI-DAYLIN, the liquid multivitamin they like just as it comes from the bottle, awaits them. With its appearance of yellow honey, VI-DAYLIN even *looks* good. And one taste convinces the most skeptical child that appearances are not deceiving where lemon-candy-flavored VI-DAYLIN is concerned.

One look at the formula shows VI-DAYLIN is also good vitamin therapy. A 5-cc. teaspoonful a day, the average dose for children up to 12, supplies *six* essential vitamins in recommended daily allowances.

VI-DAYLIN's stability at room temperature permits storage in cupboard or pantry, makes purchase of larger sizes practical. For infants, it mixes readily with milk, juices or other foods. Leaves no fishy odor in kitchen, no resistant stains on clothing. VI-DAYLIN is supplied in 90-cc., 8-fluidounce and 1-pint bottles.

Abbott

NOTE THE FORMULA

Each 5-cc. teaspoonful of Vi-Daylin contains:

Vitamin A	3000 U.S.P. units
Vitamin D	800 U.S.P. units
Thiamine Hydrochloride	1.5 mg.
Riboflavin	1.2 mg.
Ascorbic Acid	40 mg.
Nicotinamide	10 mg.